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(Continuing the Journal of the Allied Dental Societies) V. 13

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FUTURE OF THE JOURNAL OF DENTAL RESEARCH

Quotation from an announcement on page 297 of this volume. The *Journal of Dental Research* was not founded for financial profit. The *Journal* has never been a commercial project. In 1919, when the *Journal* was first issued, no dental society cared to conduct it. At the beginning we informally expressed the hope that successful performance of the *Journal's* desirable functions would soon induce at least one important dental organization to desire to own the *Journal* and to assure its permanence. Since 1919 we have repeatedly stated that the name and good-will of the *Journal* is transferable, without any pecuniary or personal reservation, to a leading dental society. Since 1929, annual reports to the American College of Dentists, by its Commission on Journalism, have stimulated the interest of several dental associations in the future ownership of the *Journal.* The Commission's public report in 1932 facilitated presentation of proposals, relating to transfer of ownership, by the (a) American Dental Association (May 22), the (b) International Association for Dental Research with the coöperation of the American College of Dentists and the New York Academy of Dentistry (May 26), and the (c) First District Dental Society of the State of New York (June 16). These three proposals are now being studied in detail by all the editors.

Quotation from an announcement on pages 521–522 of this volume. In a circular summary of the expressed views of each editor on . . . . [the above] proposals, as sent to the editors (88) after a preliminary vote in June and July and two additional formal ballots begun in September and October, respectively, it was stated (Nov. 14) that a majority of the whole board recommended that ownership be transferred to the International Association for Dental Research. . . . . The correspondence between the International Association for Dental Research and the *Journal of Dental Research*, on the problems associated with the proposed transfer of ownership and management, has been referred by President Hatton to a Special Committee of the Association for a report to the Council, which in turn will present its conclusions and recommendations at the next general meeting of the Association in Chicago, March 17–18, 1934. Until the Association indicates its final response to the above-mentioned notification, the *Journal of Dental Research* will continue under its present auspices. A statement of further developments will be published in the issue for February, 1934.

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DIFFERENTIAL RADIOGRAPHIC DIAGNOSIS IN ORAL LESIONS[1]

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I. INTRODUCTION

Correct interpretation of pathological changes, as they appear in the Roentgen picture of the mandible or maxilla, depends principally on an intimate knowledge of the physiological processes that cause decalcification or increased calcification of bone as well as absorption or new formation of osseous tissue. Different diseases produce these changes in different ways, thereby causing certain diagnostic characteristics.

II. REGRESSIVE CHANGES

1. *Osteoclasia* occurs under many conditions. It is a slow reduction of the bone, effected by resorption. It may be either physiological

[1] Presented at a meeting of the Section on Oral Surgery, First District Dental Society of the State of New York, New York Academy of Medicine, February 17, 1932. Received for publication: October 19, 1932.

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in nature, as when teeth are adjusted to the proper place in the jaw; or may be due to pathological processes caused by pressure, or elimination by absorption of infected or irritating calcified tissue. In osteoclasia, osteoclasts produce lacunae on the surface of the bone, for which reason it is called "lacunar resorption." It is also often called "erosion;" it gives an irregular outline in the x-ray picture.

2. *Osteoporosis* is another type of resorption, but occurs in Volkmann's canals and the Haversian system. It gives the cortex a typically porous appearance, often seen in chronic infection of the bone. When this process reaches a certain stage, it may result in a large perforation of the cortex by fusion of the smaller defects.

III. PROGRESSIVE CHANGES

1. *Osteogenesis* is a new formation of bone through stimulation of activity in the osteoblasts already existing, or osteoblasts newly formed. It is also spoken of as apposition. Osteogenesis of cortical bone produces periosteal hyperostosis on the outside of the bone. This is spoken of as exostosis; if the inside of the cortex is involved, enostosis, which extends into the spongiosa. Osteogenesis of the spongiosa produces a thickening of the bone trabeculae. The marrow spaces become correspondingly reduced. This is an osteoblastic activity that can also be produced by stimulation of the genetic cells, as in chronic inflammation (condensing ostitis). The spongiosa is replaced by compact bone, save for small canals containing nutrient vessels. It becomes very radiopaque, and is spoken of as sclerotic or eburnated bone. In certain deficiency diseases there may be apposition by uncalcified osteoid. This produces increased radiolucency of the bones as in rickets and osteomalacia.

2. *Calcium impregnation.* Tissue removed from the immediate influence of active metabolism attracts calcium salts. This is seen especially in semi-vital or necrotic tissue, which is the reason why a sequestrum stands out and is easily recognizable in a Roentgen picture.

IV. INVASION AND REPLACEMENT OF BONE

When bone is replaced by inflammatory tissue or neoplasm, the part of the bone containing it becomes radiolucent, and therefore

produces in the Roentgen picture a changed appearance, which is easily recognized. While it is impossible to distinguish between granulation tissue, pus, serum, or a cyst-sack in the bone, certain tumors are exceptional. Cancer tissue, as a rule, is much more radiopaque than inflammatory exudates. This, however, may be partly due to the thickness of the tumor.

The manner in which bone is attacked is generally visible in the Roentgen picture, and is often an important diagnostic factor. Pressure exerted by central lesions may produce changes, such as expansion of bone, bulging of the cortex, and pressing apart of the roots of teeth. It is also possible to tell whether a tumor invades the bone, or whether it is separated from it by a fibrous capsule.

V. MANDIBULAR JOINT

The mandibular joint is not always easily roentgenographed, especially when there is bony ankylosis. As a rule the regular mastoid position gives the best results.

1. *Dislocation.* While the dislocation of the mandibular joint has distinctive clinical indications, it is, however, at times necessary to ascertain by Roentgen diagnosis whether there is a fracture of the condyle. Upward and backward dislocation involves the roof of the glenoid fossa or the tympanic plate. Forward dislocation generally causes no injury, but the head of the condyle is moved in front of the articular eminence.

2. *Fracture* occurs generally at the neck of the condyle. Often it is necessary to have a lateral view and an A. P. view of the head to make the diagnosis. This fracture is frequently overlooked, especially if there is a more evident fracture in some other part of the mandible. The neck of the condyle is almost always displaced in a forward direction, except in a case where the blow came from the side, when there may be an oblique fracture along the posterior border of the ramus of the jaw. This results in an outward displacement of the distal part of the condyle fragment.

3. *Ankylosis.* Changes in the joint occur from infections or arthritic conditions. These may cause a roughening of the joint margin, which is sometimes demonstrable in the Roentgen picture. After more destructive suppurative processes, the healing process results

in fibrous or bony ankylosis. In the case of bony ankylosis, there is of course complete obliteration of the joint space, which again can be demonstrated roentgenographically.

VI. INFECTIOUS BONE LESIONS

1. *Ostitis.* In bone infections we see both regressive and progressive changes, depending on the reaction of the tissue to the invading organisms. *Suppurative ostitis.*—This is the active process of destruction. Both soft and hard tissue are rapidly destroyed, and a great deal of broken-down tissue accumulates as pus. The ensuing bone cavity is clearly visible in the x-ray picture. Later, when the active process has burned out and repair has set in, so-called scar bone is formed. This is sclerotic bone of great radiopacity. *Granulating ostitis.*—In the chronic type of bone infection, progressive changes slowly produce a replacement of bone by inflammatory granulation-tissue. The effect on the radiability of the bone is similar to that of the acute or suppurative type of ostitis. If the disease starts from an infected tooth, it seldom becomes more extensive than the well-known inflammatory dental granuloma. Occasionally, however, a large area of bone may be involved, which is the type we consider under this heading. The Roentgen picture shows an irregular outline and a gradual change from normal to abnormal tissue at the margin of the defect. *Condensing ostitis.*—Here the progressive tissue-changes produce new bone instead of fibrous tissue. The marrow spaces are obliterated and the resulting sclerosis is easily recognizable roentgenographically. It is generally centered around the focal point of the infection. *Residual infection.*—Long after extraction of the infected teeth, residual bone infections may be found. These are seen as radiolucent areas of diffused outline in a Roentgen picture. Generally they contain small sequestra or a spicule of the root of a tooth.

2. *Osteomyelitis.* This condition is generally accompanied by loosening of teeth, which often leads to a mistaken clinical diagnosis. The Roentgen picture in the early stages indicates a rarefied condition caused by enlargement of marrow spaces, several fusing into one. Later these spaces are joined and lengthy channels appear, and may enclose sequestra, which become more highly calcified, making them prominent in the Roentgen picture. The bone of the alve-

olar process between teeth shows a similar rarefied condition, which differentiates it from periodontitis when seen in the dental film.

3. *Syphilitic involvement of the jaw.* Secondary syphilis may affect the periosteum, but bone lesions occur more often in tertiary syphilis. The skull and mandible are frequently involved. In the mandible diffuse osteomyelitis is produced and a gumma often forms under the periosteum. This periosteal involvement differentiates the condition from osteomyelitis. It may give rise to periosteal bone-formation. In the skull and hard palate, small and large gummata may form, attacking the surface of the bone. The palatal process of the maxillary bone is often perforated, and in the skull, ostitic bone cavities are formed, often in many different sizes. Here also there is new bone-formation—in lamellae parallel to the surface of the bone. The Roentgen picture of the mandible is similar to that in ordinary osteomyelitis, except for the marked periosteal participation. The lesions in the palate and skull have the character of ostitis. In a general way a differential diagnosis is easily made by the Wassermann reaction.

VII. LESIONS OF UNCERTAIN ETIOLOGY

1. *Ostitis deformans* (*Paget's disease*) is believed to be a chronic inflammatory malady. It occurs in middle age or in later years, and is almost always multiple, the lower limbs and the skull being most frequently involved. It affects the maxilla. I have personally seen no case in which changes are produced in the mandible. Spaces are formed in the medulla or spongiosa of the bones, and filled with a fibrous tissue. New bone-formation may take place, and varies in the different cases. The overgrowth of bone sometimes results in marked increase in the size of the head, which is characteristically shown in the Roentgen picture. There are good-sized areas of decreased density in the skull with marked hypertrophy, the new bone having a tendency to split lengthwise into lamellae. There is no participation of the periosteum as in syphilis. The maxilla shows a typical rarefaction, visualized even in a dental film.

2. *Xanthomatosis* is also known as "Schüller-Christian" disease. It is caused by a disturbance of the lipoid metabolism, and occurs mostly in children. Sosman of the Peter Bent Brigham Hospital

reported several cases. I am indebted to Dr. Vogt of the Children's Hospital for the material connected with this disease. The principal symptoms which may or may not be present are exophthalmos, diabetes insipidus, gingivitis, stomatitis, and dwarfism. All the bones may be involved, but the skull and jaws are principally affected. The defects are not caused by a neoplasm, but by a phagocytic action of the reticulo-endothelial system. The tooth germs may be destroyed or displaced, resulting, in treated cases, in mal-position. The Roentgen picture shows large well-circumscribed areas of decreased density without inflammatory reaction at the edge of the defect, as in ostitis. In the skull these give, in some cases, a typical effect spoken of as "geographic skull." The teeth or tooth germs may be displaced, or may appear to be floating in space owing to the disappearance of the calcified surrounding bone.

3. *Ostitis fibrosa cystica* (*hyperparathyroidism*) attacks the entire skeleton, suggesting a general etiology. According to the latest investigations, it can now be definitely classed as a bone reaction to hyperparathyroidism, which may either be due to increased secretion, or adenoma of the parathyroid gland. It is associated with increased blood calcium and calcium output. As much as six times the normal amount of calcium may be found in the urine. Decalcification of the bones and fibrosis of the bone marrow occur, with the formation of osteoclastomas and cysts. Any part of the long bones may be involved, and the spongiosa of others. It causes a thinning of the cortex, and only a slight widening of the bone, in contrast to the benign giant-cell tumor, where there is a tremendous expansion of the bone. The bone lesions are a product of inflammation and repair. Ostitis fibrosa cystica has a tendency not to heal after operation, but repair of the bone lesion occurs if the parathyroid tumor is removed. The Roentgen appearance is quite characteristic. It shows a multicystic condition, with a thinning of the cortex. The expansion of the bone may or may not be present in the jaws.

4. *Ostitis cystica haemorrhagica* (*traumatica*). We all have seen cysts that we were unable to classify as cysts of dental origin, nor were they of the multiple type affecting the entire skeleton. The history of all such cases should be carefully examined. Often the patient remembers an accident or trauma that occurred some months

or years before. In such cases ostitis cystica haemorrhagica should be considered in the diagnosis. The lesion is probably closely related to the benign giant-cell tumor, which Codman, Konjetzny and Benecke believe are caused by intermedullary hemorrhage due to rupture of the nutrient artery. I can only present one case of ostitis cystica haemorrhagica affecting the mandible, which I have studied histopathologically. In ostitis cystica haemorrhagica a slight trauma, such as a blow or infraction, may cause a rupture of the nutrient artery. A hematoma forms in the spongiosa, causing resorption of the bone trabeculae. A cavity forms in which inflammation and repair take place. Granulation tissue is formed, encapsulating the residue of the hemorrhage, and osteoclasts are generally present in large numbers, especially along the bone. The bone cavity may become very large, but does not involve the cortex. In studying the reported cases in the literature, especially those by Schneider, I find the following characteristic Roentgen appearance: The picture of the bone cavity shows an outline corresponding more to the ostitic than the cystic type of lesion. There is no typical cortical bone-lining. The teeth may or may not be connected with the area, but they generally are not actually involved. The cortex is not greatly expanded, and there is no typical trabeculation as in giant-cell tumors or cystic adamantinoma.

VIII. FOLLICULAR CYSTS OF JAWS

Bone cysts of dental origin are classified as follicular and radicular cysts. The follicular cysts, which may be found early in life, but may remain undiscovered for years, are produced by a pathological change in the enamel organ.

1. *Simple follicular cysts.* The enamel organ, instead of forming tooth tissue, becomes a cyst, which accumulates fluid and expands the bone.

2. *Dentigerous cysts.* In this type of follicular cyst, a tooth is formed by the tooth germ, but the enamel organ forms a cyst around the crown of the tooth. As it expands, it produces pressure causing bone resorption. This pressure also exerts its influence on the occlusal surface of the tooth, forcing it to the inferior border of the

mandible or the superior part of the maxilla. Dentigerous cysts may be found on deciduous as well as permanent teeth.

3. *Cysts containing odontoma.* A follicular cyst may be formed as just described; but, instead of making a tooth, the tooth germ produces an irregular mass of soft and hard tissue. Sometimes a number of small irregular teeth are formed and held together by a cement- or bone-like structure.

4. *Multiple follicular cysts.* The Roentgen diagnosis is made from the appearance of the picture of the radiolucent area. It should have a rounded circumference, its shape being governed by the resistance it meets while expanding. It is never found at the root part of the tooth, which differentiates it from the radicular cyst. The bone shows a typical reaction, forming a lining of cortical bone around the cyst membrane. The Roentgen picture, therefore, always shows a distinct line of dense bone around the radiolucent defect. Multiple follicular cysts are due to one, two, or more adjoining tooth germs being involved in the cyst formation. They are generally separated by bony septa, and may be of the same type or a combination of different classes of follicular cysts.

IX. RADICULAR CYSTS OF JAWS

An entirely different etiology is presented by the radicular cyst. It forms from an epitheliated dental granuloma. At an early stage of development it can be distinguished from the granulating ostitis type of granuloma by the typical reaction of the surrounding bone. As in the case of the follicular cyst, a layer of cortical bone surrounds the lesion. When it expands, neighboring teeth are often involved. Frequently these teeth may only be projected into the area, when actually their location may be anterior or posterior to it. Radicular cysts may sometimes be found in an edentulous part of the mouth, having remained after tooth extraction, or formed from a granuloma left after tooth extraction. Pressure by a cyst on the nerve, especially in the mandible, may produce a paresthesia in the anterior part of the teeth and lip, similar to the anesthesia caused by the mandibular injection. In the maxilla, the nasal cavity and the maxillary sinus may become involved, and the condition is best diagnosed in a sinus picture taken from Water's position.

X. NEOPLASM

The x-ray furnishes a means of examining and studying neoplasms that can be performed without harming the patient. In many cases where one bone of the skeleton is affected, x-rays should be taken of the other bones to distinguish solitary lesions from multiple neoplasms. When making a diagnosis, the clinical findings should always be considered in conjunction with the x-ray picture. Roentgen evidence alone can seldom be relied upon, if it is necessary to make a differential diagnosis; and in many cases a microscopic examination alone will furnish a definite proof of the pathology.

1. *Neoplasms derived from tissues of the tooth germ.* When tissues of the tooth germ give rise to tumors, they usually form in the spongiosa of the bone. They may develop from epithelial tissue, such as different types of cells making up the enamel organ; or from structures derived from it, such as epithelial rests of the sheath of Hertwig. Connective-tissue elements from the tooth germ may also give rise to tumors, especially cells of the dentine-forming pulp and the cementum and the bone-forming peridental membrane. Much more rarely an epithelial tumor forms from basal cells of the mucous membrane (the region of the dental lamina); or from cystic epithelium, from which it extends into the neighboring bone. *Adamantinoma* is formed from the epithelial structures of the tooth germ. It may be cystic and present a multilocular appearance. The solid type sometimes develops into an adamantine epithelioma. The adamantinoma is more common in the mandible. In its advanced form it expands the bone extensively, and on the outside may form a swelling the size of a child's head. Enamel is never formed in an adamantinoma except when dentine is formed also, and then it should be classified as odontoma. The Roentgen picture shows the outline of the projecting growth, which in the maxilla may invade the maxillary sinus, the nasal cavity, and the pharynx. The cystic type shows the characteristic subdivisions and trabeculations, which sometimes resemble a benign giant-cell tumor. *Odontoma.*—When all parts of the tooth germs participate in the formation of the tumor, an odontoma results. Produced by faulty functioning of the tooth germ, it varies greatly in form and make-up. Sometimes it appears to be only a mal-

formed tooth; in other cases, it presents a mass consisting of two or more teeth fused together; or from the point of view of gross pathology, there may be a solid calcified structure of irregular shape. Under the microscope this formation shows a fusion of all kinds of irregularly shaped small teeth held together by osteocementum, or fibrous connective-tissue. This tumor is generally not attached to the bone. The Roentgen picture is very typical and unmistakable. The structure of the odontoma generally shows the outlines of the teeth contained in it. A dark line separating it from the bone shows the membrane limiting it, and also attaching it to the osseous structure.

2. *Neoplasm of the bone.* The most frequent tumors of the bone are the extra-periosteal fibrosarcoma and the osteogenetic sarcoma. Ewing's tumor and multiple myeloma are only rarely found in the jaws. *Peripheral fibrosarcoma* is formed from the periosteum, and pushes away the soft tissue surrounding the bone. It does not infiltrate, but frequently invades the bone. The Roentgen picture shows the bone cavity containing the fibrosarcoma; and, if the exposure is right, the outline of the tumor mass extending away from the bone is visible. *Osteogenic sarcoma* is formed in the spongiosa from the ancestors of bone-forming cells. According to Kolodny's excellent monograph on "Bone sarcoma," it is not necessarily a bone tumor that produces bone, but a tumor made up of cells having the potential ability to produce bone. Therefore, places in the bone are filled with soft parts of the tumor, while other areas show productive bone activity. The Roentgen picture has the following features in osteogenetic sarcoma: common absence of a definite outline of the tumor; structural, dense, bone formation springing from or attached to the involved part of the bone. These bone spicules or lamellae are seen in the x-ray radiating from the bone in fan-like or sun-ray fashion. Only in very rare cases is new osseous tissue arranged in layers parallel to the bone. In some cases the tumor exhibits a structureless ossification, which, in the x-ray, shows a spotty appearance of the bone owing to the presence of unossified radiolucent areas. Many roentgenologists believe that the so-called "lipping" of the periosteum is a typical feature of the malignant tumor. "Lipping" is absent from benign osteogenic growth. The osteogenic sarcoma presents a different appearance at various stages of development. In

the early stages it may be mistaken for chronic osteomyelitis tending toward sclerotic changes.

XI. CARCINOMA

The primary lesion may arise from the oral epithelium, but more frequently carcinoma invades the bone from a primary lesion in the lip, the cheek, the tongue, or the mucous membrane of the floor of the mouth. We classify carcinoma according to location, as central or intra-osseous, infiltrating or extra-osseous, and metastatic types.

1. *Intra-osseous or central carcinoma.* Epithelial rests from the enamel organ may give rise to carcinoma either directly, or through derivative pathological structures, such as epitheliated granuloma, cysts, and adamantinoma, especially the solid type. In the maxilla, carcinoma may also arise from the membrane of the maxillary sinus or the nose.

2. *Extra-osseous or infiltrating carcinoma.* Carcinoma of the cheek, palate, floor of the mouth, or tongue may frequently infiltrate the bone, and in advanced stages it is often impossible to say in which part the tumor originated. The bone is generally completely destroyed, but the tumor sometimes produces necrosis owing to interference with the blood supply, and a secondary infection may complicate the picture. The Roentgen examination, in either intra- or extra-osseous carcinoma, shows extensive loss of bone. The tumor tissue, which invades and replaces the bone, is however not as radioparent as inflammatory granulation tissue. On this account, and perhaps also because of the thickness of the tumor owing to lateral expansion in very advanced cases, the Roentgen picture of the involved area has a typical quality. Instead of a very dark shadow as seen in cysts or ostitis, there is an opaque grayish Roentgen appearance. Necrosis of the cortex, especially at the inferior border, is not uncommon, and there may be a break in its continuity. Otherwise the outline of the bone is not disturbed. In the maxilla, the maxillary sinus may contain a large part of the tumor, causing either a swelling or destruction of the bony walls of the sinus.

3. *Metastatic carcinoma in the jaw.* So-called secondary carcinoma is frequently found in bones and is generally due to the glandular type. The jaws are rarely affected by it. The skull is frequently involved.

I have seen one case of metastatic carcinoma of the mandible. In this case the Roentgen picture showed osteolytic defects. In other cases, especially carcinoma of the prostate metastatic to the jaw, progressive bone changes are more common.

XII. CASE REPORTS

A. Mandibular joint. (*1*) Mr. R. C. P. Roentgen picture shows fracture of condyle with forward displacement. (*2*) Miss H. Oblique fracture of head of condyle from lateral blow. (*3*) (Courtesy of Dr. Kazanjian). Forward dislocation of condyle. All teeth extracted under local anesthesia; last, about three weeks ago. Lower jaw gradually began to protrude. (*4*) Miss S. T. Age 7. Always had difficulty in opening mouth. Underdevelopment of mandible and bi-lateral ankylosis. (*5*) Mr. O'H. Complete bony ankylosis; due to arthritis.

B. Infectious bone lesions. Suppurative ostitis.—(*6*) For three weeks swelling on face, eye closed. Infection quieted down lately; free pus discharge from fistula. *Granulating ostitis.*—(*7*) Mr. W. I. D. Previous treatment: central incisor extracted; wound keeps discharging. Lateral incisor treated without success. Roentgen diagnosis: granulating ostitis. (*8*) Mr. MacM. Suffering for weeks from infection in lower jaw; unable to receive proper treatment. Roentgen diagnosis: ostitis involving second and unerupted third molar. *Ostitis due to actinomycosis.*—(*9*) Mr. I. M. B. Infected lateral incisor, with fistula discharging pus. Roentgen examination: radiolucent area of ostitic type. Microscopic examination: fungus actinomyces. *Condensing ostitis.*—(*10*) Mr. W. History of severe attack of acute abscess. Roentgen examination: sclerotic bone surrounding root-infected apices. (*11*) Age 28; tooth treated twelve years ago for acute abscess. Roentgen picture: area with sclerotic bone. *Osteomyelitis.* —Three cases of hematogenous osteomyelitis, all with negative history as to accident and infection; teeth sound. (*12*) Never any swelling nor pain until two weeks after x-ray examination. (*13*) When first seen, swelling and pain in chin. (*14*) Tuberculosis fifteen years previously; lost 15 pounds last few weeks. (*15*) Mr. H. B. Acute alveolar abscess on left inferior cuspid. Jaw and face swollen. Two days after extraction, felt very ill; temperature 105. Examination: teeth loose. Roentgen examination: extensive rarefaction of mandible, with sequestrum formation. Diagnosis: osteomyelitis (*fig. 1*). (*16*) Miss L. Extensive osteomyelitis of mandible; shown in series of slides. Disease started from acute infection of lower tooth. (*17*) Osteomyelitis: anterior part of mandible. (*18*)

Localized osteomyelitis: anterior part of maxilla. (*19*) Osteomyelitis, with highly calcified sequestra. (*20*) Low-grade chronic osteomyelitis, with sclerosis. *Syphilis.*—(*21*) Mr. H. History of syphilis and tabes dorsalis. Large periosteal gumma formation. Large sequestrum forming. (*22*) Mrs. G. Swelling on palate and soreness of teeth for some time. Under treatment by physician. Roentgen examination: ostitic destruction of palatal process. Diagnosis: gumma with perforation. (*23*) Mr. W. Extensive osteomyelitis of mandible, with difficult differential diagnosis owing to many dental infections. Wasserman test positive. (*24*)

FIG. 1. (CASE 15.) OSTEOMYELITIS OF MANDIBLE

Woman, age 50; extensive ostitic areas in skull and forehead due to gumma (*fig. 2*).

C. Lesions of uncertain etiology. Ostitis deformans.—(*25*) Mrs. W. S.; age 61; bowing of legs, head enlarged. Roentgen examination in slides: marked involvement of skull, ilium, upper part of femori, tibia, and fingers. Diagnosis: Paget's disease. (*26*) (Courtesy of Dr. Harold Kent.) Suffering from Paget's disease. Typical porosity of alveolar bone. *Xanthomatosis.*—(*27*) (Courtesy of Drs. M. C. Sosman and E. C. Vogt.) Age $4\frac{1}{2}$; complained for six months of sore mouth and gums. Lost weight, although received cod-liver oil and orange juice. Roentgen examination: several

circular defects in skull, with sharply outlined ragged edges. Mandible shows large radiolucent areas in bone below teeth and in ramus (*fig. 3*). (*28*) and (*29*) Similar cases of xanthomatosis. *Ostitis fibrosa cystica.*—(*30*) (Courtesy of Dr. P. Butler.) Complained for several months of pains in legs, arms, and sacro-iliac region. Dentist claimed teeth affected by py-

FIG. 2. (CASE 24.) SYPHILITIC CHANGES IN SKULL

orrhea in lower jaw. Roentgen examination (Boston City Hospital): areas of decalcification in ribs, ilium, many of long bones, skull and mandible. Specimen from tibia studied; blood calcium 14 mg. per 100 cc. Diagnosis: ostitis fibrosa cystica; due to hyper-parathyroidism (*fig. 4*). (*31*) and (*32*) Cases of gen. ostitis fibrosa cystica. *Localized ostitis fibrosa cystica hemorrhagica* (traumatica).—(*33*) Age 35; kicked some years ago

while playing football; swelling of jaw. Roentgen examination: large cystic area in ramus and posterior part of mandible. Tissue-lining cystic cavity is granulation tissue; chronic inflammation, but no epithelium present.

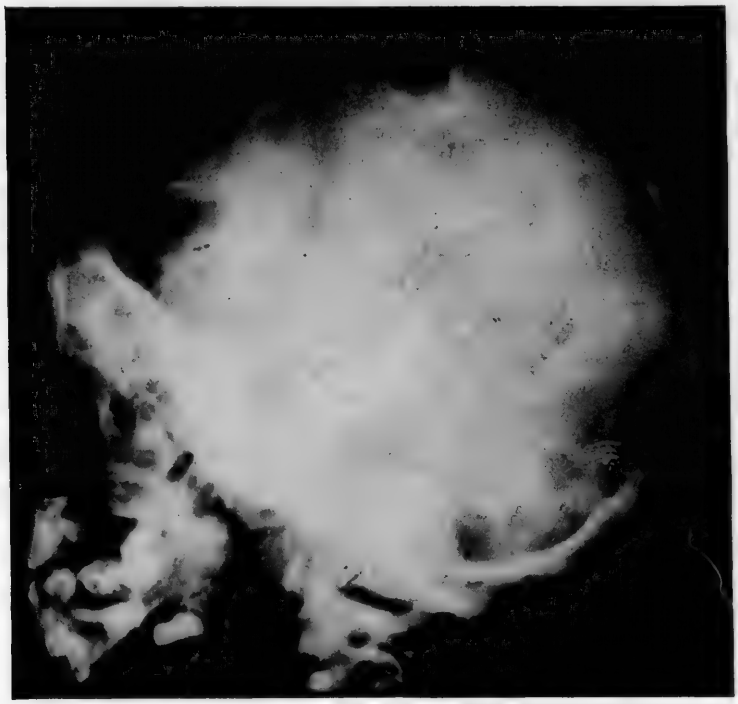

FIG. 3. (CASE 27.) XANTHOMATOSIS; SKULL AND MANDIBLE AFFECTED
(Courtesy of Dr. E. C. Vogt)

D. Cysts of jaws. Simple follicular cysts.—(*34* and *35*). *Dentigerous cysts.*—(*36 and 37*). *Cysts with odontoma.*—(*38*) Master W. Age 16; swelling under lip. Treated by dentist: pulp from lateral incisor removed. Roentgen examination: typical follicular cyst, with odontoma. *Multiple follicular cyst.*—(*39*) In mandible.

E. Radicular cysts. (*40–49*) Varied cases of radicular cysts. (*50–52*) Cysts in maxillary sinus.

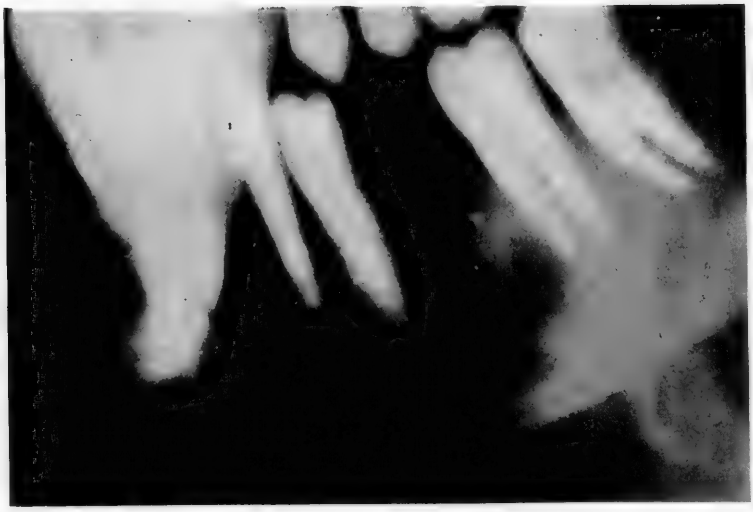

FIG. 4. (CASE 30.) OSTITIS FIBROSA CYSTICA (HYPER-PARATHYROIDISM); MANDIBLE

FIG. 5. (CASE 64.) EWING'S TUMOR OF MANDIBLE
(X-ray by Dr. George W. Holmes)

F. Neoplasm. Adamantinoma.—(*53*) R. W. Age 19; received hard blow on jaw when one year old. Slow painless growth appeared; for several years, size of orange. (*54*) Presented swelling inside of mandible. Provisional x-ray diagnosis: carcinoma. Pathological diagnosis: adamantinoma. (*55*) Mrs. W. J. C. (Courtesy of Dr. L. M. S. Miner.) History since 1888, when first operated on. Diagnosis: adamantinoma. Confirmed by pathological diagnosis. *Odontoma.*—(*56* and *57*) Geminated composite odontoma. (*58* and *59*) Compound composite odontoma. *Fibrosarcoma.*—(*60*) (Courtesy of Drs. Miner and Kent.) Age 7; large swelling at angle of mandible for several months. Roentgen examination: hard tumor;

FIG. 6. (CASE 70.) EPIDERMOID CARCINOMA INFILTRATING MANDIBLE

seems firmly attached to angle of mandible. Pathological report: fibrosarcoma with irregular trabeculae of bone. *Osteogenic sarcoma.*—(*61*) Age 36. Roentgen picture: "lipping" of periosteum and spotted appearance of mandible, characteristics of osteogenic sarcoma. Diagnosis: verified by pathological report. (*62–63*) Other cases of osteogenic sarcoma. (*64*) Ewing's tumor of mandible (*fig. 5*).

G. Carcinoma. Intra-osseous carcinoma.—(*65*) Mrs. M. McC. (Courtesy of Dr. Kent.) Age 59; no previous illness. Teeth removed three years ago. Never wore plate. Swelling appeared in mandible few months ago. Roentgen examination: large cystic cavity and loss of bony archi-

tecture of mandible. Microscopic examination from cyst cavity: epidermoid carcinoma. (*66*) Mr. J. C. O'B. (Courtesy of Dr. Fred. Cotton.) Age 55. Roentgen examination: cystic expansion of mandible. Cortex destroyed, pointing to malignancy. Pathological diagnosis: slow-growing type of cystic epithelioma. (*67*) Mrs. G. Swelling inside of mandible for some time; increasing rapidly last few weeks. Enlarged heart with mitral murmur. Poor operating risk. Roentgen examination: characteristic appearance of carcinoma. Patient died three months later from pulmonary metastasis. (*68*) Mrs. W. P. Several teeth extracted; now swelling of upper jaw. Sinus normal on transillumination. Roentgen picture: solid tumor in anterior part of maxilla extending into maxillary sinus. Pathological diagnosis: epidermoid carcinoma (Grade 1). (*69*) Miss L. Age 82. Consulted regarding ulceration behind maxillary incisor teeth. Feeble old lady; had poor heart. History of operation for carcinoma of breast. Roentgen diagnosis: carcinoma of upper jaw, operation inadvisable on account of general health. Next few months tumor rapidly increased in size. Biopsy examination showed carcinoma. Patient died, without suffering, ten months later. *Infiltrating carcinoma.*—(*70*) Mr. C. J. Age 66; five months ago six loose teeth extracted; few weeks ago, three more—jaw then somewhat swollen. Examination: hard tumor extending from incisor region to ramus, pushing tongue away. Enlargement of cervical glands. Pathological examination: epidermoid carcinoma (*fig. 6*). (*71*) Epidermoid carcinoma of lip, with infiltration of mandible. (*72*) Adenoma of parotid gland, with invasions of ramus. *Metastatic carcinoma.*—(*73*) Mr. H. T. Age 71; carcinoma of prostate gland. Roentgen examination of jaw: large radiolucent area in mandible; numbness in lip. Diagnosis: metastatic bone lesion, due to carcinoma. No pathological examination.

XIII. CONCLUSION

In studying disease, it must be remembered that the Roentgen examination does not give a picture of the pathological process, but only a record of the effect of the disease on the bony structure. More can be learned from serial examination. Roentgen pictures taken at intervals are extremely interesting and instructive, and give an opportunity to follow a pathological process through its different stages of development. In making a diagnosis from a Roentgen picture, it is always well to compare the affected bone with the normal one on the other side. Great care must be taken so as not to interpret, as patho-

logical changes, shadows caused by the tongue, pharynx, hyoid bone, or the vertebrae. It also should be remembered that the typical textbook illustrations are generally selected cases, and often show the disease in the most advanced stage with extensive tissue involvement. In many atypical cases some of the finer diagnostic indications are missing. In these cases a microscopic confirmation is often necessary before a final diagnosis. The Roentgen examination often gives other valuable information not available in any other way, such as the extent of the involvement of the bone; whether there is infiltration of the neighboring tissue; and what relation the lesion has to neighboring structures. It is necessary for the clinical diagnostician and the pathologist and the roentgenologist to work together, not only for the good of the individual patient, but also for the advancement of the science of diagnosis of the lesions of the maxillary bones.

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XV. DISCUSSION

A. L. Greenfield, D.D.S. (New York City): We have listened to an extremely excellent discourse on this very important phase of diagnosis. Dr. Thoma ably sounded the keynote of this paper when he said: "Correct interpretation of pathological changes, as they appear in the Roentgen picture of the mandible or maxilla, depends principally on an intimate knowledge of the physiological processes that cause decalcification or increased calcification of bone, as well as absorption or new formation of osseous tissue. Different diseases produce these changes in different ways, thereby causing certain diagnostic characteristics." Many points as brought out are so complete that they require no further discussion. Some, however, owing to their extreme importance, may need reiteration and further discussion.

Foreign bodies in the mandible and maxilla. It is extremely difficult, sometimes impossible, to differentiate radiographically between a root fragment and sclerotic areas in bone. This is particularly true where the root apex has been broken off obliquely and not transversely. When this happens, we do not see the regular, sharply defined, clear-cut radiopaque area in the bone that is so necessary for diagnostic differentiation between a root and sclerotic bone. In the radiograph we also see cases of old encapsulated roots. They frequently appear so irregular in outline that, here too, radiographically one cannot with any degree of certainty make a differential diagnosis.

In making a diagnosis of a root in the antrum, great care must be exercised. The radiograph lacks depth or perspective. Very frequently the roots of a tooth straddle the antrum and radiographically appear to penetrate the antrum. If the apex of such a tooth is fractured, a subsequent erroneous diagnosis of a root in the antrum might be made. Of course, if the peridental membrane and lamina dura are seen surrounding the root apex, we can definitely conclude that the root is not in the antrum. If these supporting, or rather surrounding, structures are not visible, then there still exists the possibility that the root may have been pushed along under the periosteum and not be in the antrum. A postero-antero view of the maxillary sinus will aid us here. If, in such a film, the root appears within the confines of the antrum, we can make the definite diagnosis that the root is in the antrum. But if the root is not found in the antrum, in a postero-antero view, it may be in the antrum nevertheless. The apex may be so small, or so close to the walls of the sinus, that it is not visible on the film. However, if it does appear in the sinus, a fairly conclusive diagnosis can be made. If the root has been pushed under the periosteum, the case must be differentiated clinically, not radiographically.

Residual infections. These areas of infection are usually easily noted and recognized in the radiograph. They are seen as diffuse radiolucent areas in the bone. If, as described by the essayist, they contain a small spicule of root or a sequestrum, the diagnosis is more easily made. However, I wish to bring out the fact that, in some cases, radiolucent areas in edentulous parts are not necessarily areas of residual infection. If care is taken, faint strands of cancellous structure can be seen in this radiolucent area. In my opinion, loss of stimulation of the bone, owing to loss of the tooth, prevented complete normal regeneration of the bone in these cases.

Osteomyelitis. It is well to remember that for a short period of time no destruction of bone takes place in osteomyelitis. The infection is, at first, confined to the medullary canal and within the haversian canals. These

canals are filled, but no destruction has taken place. Therefore, early in the progress of osteomyelitis there is a period during which radiographically the bone appears normal. The patient may exhibit all the symptoms of osteomyelitis, with extreme swelling and inflammation of the soft tissues surrounding the bone, extreme pain and high temperature; and the radiograph may appear normal. This is due, as was said previously, to the fact that as yet there has been no bone destruction. A similar condition is seen in an acute alveolar abscess, when the abscess is acute from the outset—not a chronic abscess that flares up and becomes acute. In acute osteomyelitis we first see radiographically a diffuse rarefaction in the bone. Later, black channels are seen, surrounding areas of normal bone, which eventually become sequestra.

Here it is well to digress for a moment, as an important point in differential diagnosis is brought out. Baetjer and Waters of Johns Hopkins say: "This is an important point of differential diagnosis, because every pathological process in bone must be viewed as malignancy until proven otherwise. Malignancy starts from one central point and radiates equally in all directions, absorbing the bone as the growth advances, but never appears as separated areas with normal bone in between, and never produces sequestra. The osteomyelitic infection will finally pierce the cortex in one or more places, leaving areas of normal cortex between them. Here again we have another important point of differential diagnosis, as in malignancy where the growth reaches the cortex; it destroys it completely as a whole, and not in parts as osteomyelitis does." After the cortex has been pierced, nature attempts to limit the destructive process, which is accomplished by building up a new bony wall at the edge of the infection. Since the infective process has reached the periosteum, deposits of bone will be seen on the outside of the cortex. In chronic osteomyelitis this deposit might be so extensive as to give the bone an irregular shape and make it appear expanded or thickened. However, it is well to remember that osteomyelitis does not expand the cortex.

To summarize, acute osteomyelitis shows bone destruction with but little bone formation. In chronic osteomyelitis we have excessive bone production with but little bone destruction. At the beginning the x-ray may fail to show any bone destruction. We then see a diffuse rarefaction in the bone. Then black channels are seen surrounding areas of normal bone, which later become sequestra. In the chronic cases we see bone production that, in the radiograph, appears as dense, homogeneous, compact masses in the bone. They are laid down on the outside of the cortex, and the bone appears irregular and thickened.

Syphilitic osteomyelitis. The radiograph in such cases may show a picture varying from a periostitis to an advanced osteomyelitis. In luetic osteomyelitis, the periostitis is marked—calcium salts are laid down more or less parallel to the bone. This type of osteomyelitis may produce the same bone changes as the inflammatory type, and sometimes cannot be differentiated. However, in syphilitic osteomyelitis frequently more than one bone is involved. Another point in differential diagnosis is that the clinical picture does not harmonize with the radiograph. In syphilis the film may show destruction to such an extent that one would expect a very inflamed, tender part with accompanying fever. Instead, clinically we find only slight tenderness, no fever, and very little or no inflammation. A Wassermann test, or preferably an examination of the spinal fluid, will aid in the diagnosis.

Osteitis deformans (*Paget's disease*). As described to us by the essayist, this disease is believed to be a chronic inflammatory one, occurring later in life, the average age being about fifty years. The pelvis, femur, and tibia are most frequently affected; the spine least. The changes produced in the skull are most typical. In the long bones the radiopaque and radiolucent areas appear as longitudinal striations. This is the result of rarefying and proliferative osteitis. In the skull, round knoblike masses will be seen lying between the outer and inner tables of bone; sometimes, on the outer table. The maxilla may be affected, showing the characteristic increase in size, and the characteristic rarefaction and radiopacity of the bone.

Osteitis fibrosa cystica. Dr. Thoma's very excellent description of the *generalized* osteitis fibrosa cystica needs no further discussion. Drs. Jaffe, Bodansky, and Blair have definitely shown that the injection of parathormone into a guinea pig produces this disease. It is well to remember that the greatest amount of resorption takes place at the sites of most active growth of bone or bone formation, such as in the long bones. The lower jaw also shows extensive decalcification and resorption. In the regions of less active bone growth, a simple thinning of the bone is sometimes the only change observed. We must, however, discuss the *localized* osteitis fibrosa cystica. The etiology is not definite, and it is thought to be due to some trophic disturbance. In these cases the condition is confined solely to the jaw, no other bones being involved. In both the localized and general cases of osteitis fibrosa cystica, the pathological picture is bone destruction and bone production. Fibrous tissue forms, causing destruction of the bone. The fibrous tissue may subsequently undergo cystic degeneration. Radiographically these localized areas of osteitis fibrosa cystica may appear in one of several ways:

(a) A circumscribed radiolucent area, in which we see irregular masses of bone.

(b) We sometimes see only the radiolucent area.

(c) It may appear as a single cyst, separated into numerous small cysts by heavy radiopaque septa. The cortex is intact; it may be thinned out or show considerable expansion.

Secondarily to osteitis fibrosa cystica due to trauma, an internal hemorrhage may occur, encapsulation take place, and a hematomcyst be produced. In such cases, microscopically, we can see, in the region or neighborhood of the hematomcyst, the characteristic changes of osteitis fibrosa cystica. This hematomcyst (secondary to an osteitis fibrosa cystica) was described by Dr. Furedi, who also described a hematomcyst that developed in *previously healthy bone.* This was also discussed by Dr. Theodor Blum in a recent article entitled "Unusual bone cavities." In this type of hematomcyst, we start with normal bone in young people. Some form of trauma causes an internal hemorrhage, with all the subsequent sequelae and the final production of a traumatic hematomcyst. Unlike the hematomcyst that occurs secondary to osteitis fibrosa, the traumatic cyst developed in previously normal bone shows *no* characteristic changes of osteitis fibrosa cystica. Radiographically these traumatic cysts appear as well defined radiolucent areas without the characteristic white line about the periphery (seen about a radicular or follicular cyst). The aforementioned article states that "wherever the teeth apparently are involved in the bone cavity, the exposed root is always covered by a distinct peridental lamella."

While on the subject of bone cavities, it will not be amiss to talk about cavities (seen as radiolucent areas in bone) that are normal. Very frequently, owing to a peculiar arrangement of the cancellous bone, an area is seen similar to a cyst. The condition is usually bilateral; a radiograph taken of the other side discloses the same condition.

The description of the cysts of dental origin is so complete that no further discussion is indicated. One point bears repetition; namely: when the cyst is seen in the radiograph extending to the neighboring teeth, it does not necessarily involve those teeth. The cyst might be buccally or lingually to the teeth, and these organs will appear projected into the cyst cavity. In the paper, Dr. Thoma stated radicular cysts "form from an epitheliated dental granuloma. At an early stage of development it can be distinguished from the granulating ostitis type of granuloma by the typical reaction of the surrounding bone." There has been some question as to the correctness of this view. Some men claim that epithelial cells may be found in both these types of granuloma, and therefore both might develop into the radicular cyst.

Adamantinoma may be cystic or solid. The cystic adamantinoma, or multilocular cyst, usually shows in the radiograph as one large cyst together with several small ones. The solid adamantinoma presents a picture that is sometimes extremely difficult to differentiate from a cyst. It appears as a radiolucent area with an extremely thick, heavy, irregular white outline about it. From the white line, small bony projections extend into the mass.

Extra-periosteal fibrosarcoma. These tumors lie next to the bone. They do not infiltrate it, although they may cause absorption on the adjacent surface of the bone. In short, they produce a more or less contour-deformity. Codman says that they appear less likely to metastasize than osteogenic sarcomas. As Dr. Thoma stated: "the Roentgen picture shows the bone cavity containing the fibrosarcoma; and, if the exposure is right, the outline of the tumor mass extending away from the bone is visible." An important point to be advanced in conjunction with this statement is the fact that this cavity or indentation may be on the buccal or lingual surface of the bone. Since this contour-deformity might be very slight, it may not be visible in the radiograph ordinarily taken. It is best to take a bite film, directing the rays from below upward, through the floor of the mouth, at right angles to the film. This will show the cavity if present, and may give a clearer outline of the tumor.

The right-angle bite-film technic is also useful in cases of benign giant-cell tumors located in the lower jaw. On account of the density of the plates of bone, the characteristic radiopaque trabeculations may not be visible in the usual radiograph. The bite-film shows this clearly; reveals the extent of expansion of the cortex; and also discloses more readily any absorption of the cortex. In fact, a bite-film is indicated in all cases of pathological conditions involving particularly the mandible.

Osteogenic sarcoma commonly shows a considerable amount of bone production, usually radiating outward in striations perpendicular to the bone. We may also see types that simply destroy bone, and therefore do not show the characteristic radiating spicules. This type occurs rarely. Osteogenic sarcoma nearly always shows radiographically both destruction, affecting the cortex and body of the bone, and the characteristic bone production.

Conclusion. While the radiograph is a marvelous aid in diagnosis, it should not be exclusively depended upon. It constitutes a considerable portion of our diagnostic data, especially when it shows a characteristic picture. A really accurate diagnosis of the case can be accomplished only by the close coöperation of the roentgenologist, clinician, and microscopist.

SOME OBSERVATIONS ON ENAMEL DEVELOPMENT AS SHOWN IN THE MANDIBULAR INCISOR OF THE WHITE RAT[1]

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The study of the development of enamel, especially that part concerned with the actual hardening of the tissue, presents considerable difficulty when attacked by the usual biological methods. Perhaps because of this situation there is a rather general disposition to accept a theory of finished enamel-rod production by the individual ameloblast. However an exception must be credited to Dr. Theodore Beust (1), who, in 1928, voiced a vigorous protest against this theory. Beust studied the intrafollicular condition of the enamel layer in pig material. He observed that only the tips of the cusps of the enamel-cap were hard enough to be recognized as enamel, while the remainder was of such a nature that it could be easily removed with the finger nail. This indicated some very essential mass-differentiation in developing enamel.

In making celloidin sections of decalcified, unerupted teeth of young pigs, I noted that there was a consistent loss of a part of the enamel layer. Where the tooth was older, the loss was greater. In all the cases observed, the junction between that which remained and that which had been lost was not a tapering off, but an abrupt termination. *Figs. 1* and *2* illustrate this condition. The same thing was noted in the enamel layer of sections of decalcified mandibular incisors of rats. Here the enamel tissue in the incisor terminates in an area approximately beneath the first molar. This condition is shown in *fig. 3*. Mrs. Mellanby (2), in her work on the effect of diet on the teeth of dogs, noted that in ground, stained sections of puppy teeth, "secondary

[1] Read at the tenth general meeting of the International Association for Dental Research, Columbus, O., March 19–20, 1932; *Journal of Dental Research*, 1932, xxi, p. 422; June.

calcification" (which did not take the stain) did not occur until the practical completion of a primary state that took the stain. Since the ordinary decalcification process results in a disappearance of well-calcified enamel, this condition, which has been described in pig and rat material, indicates the abrupt termination of a well-calcified condition, and points to the process of calcification as a mass action involving a considerable area of completed organic matrix.

There is another method of attacking this problem. It is by a study of ground sections in which the organic and inorganic materials of a continuously-growing enamel-layer, such as is found in the rat incisor, are preserved. One must use a method of observation whereby the advent of crystal forms in this growing enamel-layer may be seen. The polarizing microscope offers an opportunity for such observation. The technique, simmered down to its essentials, is a matter of demonstrating crystal forms—and the absence of the same—in the enamel layer, in progressive stages of formation. Details of this technique have been given in a previous study of tooth tissues (3). It will suffice to state here that the crystals of which enamel is composed are anisotropic, and hence demonstrable in polarized light. They have been determined to be isomorphic with those found in apatite, which are hexagonal in shape and hence in the uniaxial group.

Let us attempt to visualize what we should be able to see, in the enamel of the rat incisor, with polarized light, in case the theory of the formation of the complete, calcified enamel-rod by the individual ameloblast is true. In the first place, we should find that the dentinal end of rods are crystalline, while the portion near the ameloblast is free from crystals. *Fig. 4* is a diagrammatic illustration of such a condition. If the process of crystallization passes *through* several optically different phases, as there is reason to believe it does, then these phases should show in individual rod development. In the second place, the junction between the crystalline and non-crystalline parts of adjacent rods should form a slanting line of considerable length, from the bare beginning of crystal structure in a rod to the fully crystallized rod. The condition in decalcified sections did not show this to be the case. And last; if periodicity is a natural function of enamel calcification, for such is the present explanation of the lines of Retzius, we should be able to see alternating conditions of complete and incomplete crystallization in each rod.

With this picture in mind, let us proceed to the examination of the rat mandibular incisor enamel-layer. *Fig. 5* illustrates a ground section of one side of a rat mandible including the length of the incisor. We first examine a portion of the enamel layer in the region near the root of the incisor, just posterior to a line dropped from the distal of the second molar. In *fig. 6* such an area is shown as it appears in ordinary light. With polarized light there is no evidence anywhere in this area of any crystalline material, and a photograph under this condition would show nothing but a dark field. Although the enamel layer at this point is practically as wide as in the completed enamel, there is no sign of any calcification even at the dentinal end of the rods. We are evidently dealing here with a large area of non-crystalline material. A thin section in this area exhibits a number of ladder-like structures, assembled side by side with the spaces between the rungs of the ladders holding rather elliptical sections of material. If we were to reconstruct a three dimensional figure from a series of such sections, the result would be an association of septated tubes with somewhat flattened pea-shaped bodies occupying the tubes between the septa. That these bodies, or globules, are not very securely attached in place might be deduced from the fact that they are missing from places on the section where the tubes have lost a part of the side wall. These globules are the fore-runners of the enamel discs described by Williams. It is to be noted, however, that they have been produced here in great numbers over a wide area, without any sign of crystallization, even near the dento-enamel junction.

There is no sign of crystal formation in any part of the enamel-layer until we reach a point which lies approximately below the first-molar tooth. As this area is entered, we get the first intimation of crystal formation. But strangely enough, it is not confined to the part of the enamel near the dento-enamel junction nor is it in the globules. Instead—it is in those long areas described above as the sides of the ladders, and pretty well throughout the length of these structures, extending from a short distance from the dentin to a short distance from the ameloblast layer. That it does not reach completely from the dentin to the ameloblasts is due to the fact that, in the enamel of the rat incisor, there is quite an abrupt change in rod direction at these places. The greater part of the rod lies in the plane to which we have

ground our sections, and it is the large central area of the enamel layer to which we shall confine our observations. This is necessary because, in the use of this technique, one must be able to orient the long axis of the rods with certain features of the microscope. The change in direction at the ends of the rods in no way invalidates the data secured, provided we recognize the import of the conditions. The crystallization phenomenon, shown under crossed nicols so suddenly throughout the area below the first molar tooth, presents an alternating condition of bright, or birefringent, and dark lines. The birefringent lines are the crystallized areas and, as has been noted, are the sides of the ladder-like structures. The dark lines are the rows of globules. *Fig. 7* is a photograph of this condition under polarized light. *Fig. 7a*, the same area in ordinary light, shows the impossibility of recognizing the crystalline condition without the polarization of the light rays.

As we proceed forward on the incisor enamel, the condition shown in *fig. 7* persists for about the width of the first molar. It shows less birefringence at the start, increasing toward the center of the area, and decreasing after the centre is passed. It is an initial stage or phase of crystallization. It is characterized by crystals with a negative sign of elongation. By this is meant that, of the two separate and distinct indices of refraction of the individual crystals, the one with the higher index of refraction lies at right angles to the long axis of the enamel rod. Because of the relatively low birefringence of these thin lines, it has been impossible thus far to secure a definite interference figure on this material; and hence impossible to state absolutely the optical sign of the crystals in this initial phase of calcification. However, I believe we may be permitted to reason by analogy, in the absence of this one determination, since we have others to base our deductions upon. The information secured from a previous study (3) of human enamel, and the fact that the signs of elongation in the phases occuring in rat enamel are reversed from those determined for human enamel, and also that the final phase in rat enamel is an optically negative crystal, all suggest that the optical sign of the crystal in the initial phase of rat enamel is positive. A negative sign here would mean a subsequent shift of 90 degrees in the crystallographic axis of the individual crystal to reach the final crystal phase, which is hardly likely. We admit the insufficiency of this type of reasoning, but it is the best we can do until

an interference figure can be secured, perhaps through the use of a larger tooth, e.g., rabbit.

Proceeding forward to an area beginning approximately at a point lying below the anterior edge of the first molar, we note another crystal change. The incisor enamel-layer at this place loses its birefringent lines. The loss is apparent first near the dento-enamel line, and the narrow dark area forms an obtuse angle with the dento-enamel junc-

FIG. 1

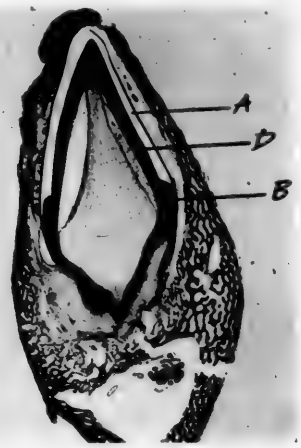

FIG. 2

FIG. 1. Section through decalcified, permanent incisor of 1-day old pig, showing complete loss of enamel layer at *A*; remaining enamel layer at *B* and *C*. Note lack of tapering-off between these areas. *D*, dentin.

FIG. 2. Section through decalcified, erupting tooth of pig, showing still greater loss of enamel layer in space marked *A*; remaining portion of enamel layer at *B*. Note again abrupt termination between *A* and *B*. *D*, dentin.

tion on the side toward the root of the incisor. From the crystal standpoint, this is a transition area, and is due to the addition of some substance to the crystal that equalizes the two hitherto different indices of refraction, causing the loss of birefringence. This addition soon exceeds the point of index equality, the isotropic condition; and we note that, entering in the same fashion as the isotropic condition did, there is a second condition of birefringence of a higher order than the first and yielding a distinct interference figure. This interference

figure is one of entering and emerging hyperbolas such as one observes when looking down on the optic normal of a uniaxial crystal. The line separating the entering and emerging hyperbolas, during clock-

FIG. 3. Section through decalcified mandibular incisor of rat. Ganoblast layer (*G*), and remaining portion of enamel (*B*), pulled away from dentin (*D*). Abrupt loss of enamel layer apparent; starts in region of incisor, lying below first molar tooth. *A* marks region in which enamel layer has disappeared.

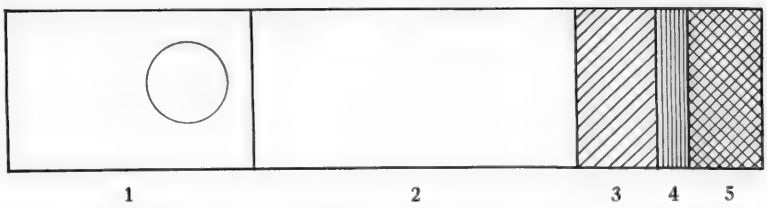

FIG. 4. Diagrammatic illustration of what might be expected if ganoblast were individually concerned in production of finished enamel-crystal phase.
1—Ganoblast. *2*—Organic (no calcification). *3*—Initial stage of calcification. *4*—Intermediate stage of calcification. *5*—Fully calcified. (Dentin-end of rod at right.)

wise rotation of the specimen, indicates the optic axis of the crystal. In this manner we determine that the long axis (optic axis) of the indi-

vidual crystals lie at right angles to the long axis of the enamel rod. The use of the selenite plate in the optical system of the microscope shows that the ray with the high index in the crystal form lies parallel to the long axis of the enamel rod. This means that the sign of elongation is positive and the optical sign of the crystal is negative. This is the final phase of the crystal form of rat enamel.

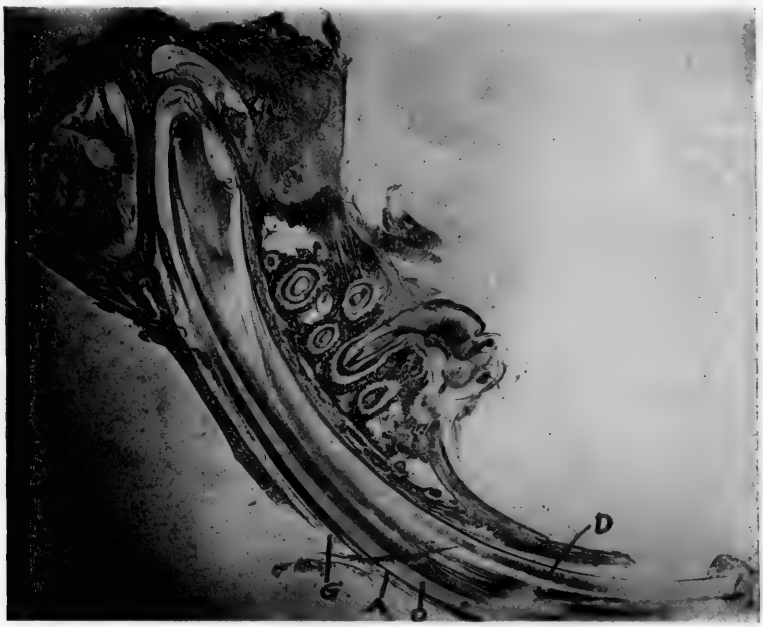

FIG. 5. Longitudinal, ground section of rat mandible, showing extent of incisor. Remaining figures were taken at higher magnifications in various portions of such sections. *A* indicates enamel layer, which can be followed well back into growing portion of incisor. *G*, ganoblast layer. *D*, dentin. (Photographed in ordinary light.)

The appearance of an initial phase of crystallization, followed by an isotropic condition that in turn is succeeded by a final phase, is visible evidence of a change in the crystals constituting the enamel rods. In changes from young to old bone, there is reason to believe that the difference is due to an initial condition of phosphate predominance succeeded by a condition of carbonate predominance (4). This may

well be the chemical change we have demonstrated optically in enamel. With the aid of a grant from the American Dental Association we have retraced the work done on human enamel (3), and in addition endeavored to determine the chemical difference between optically positive and optically negative crystals similar to those found in

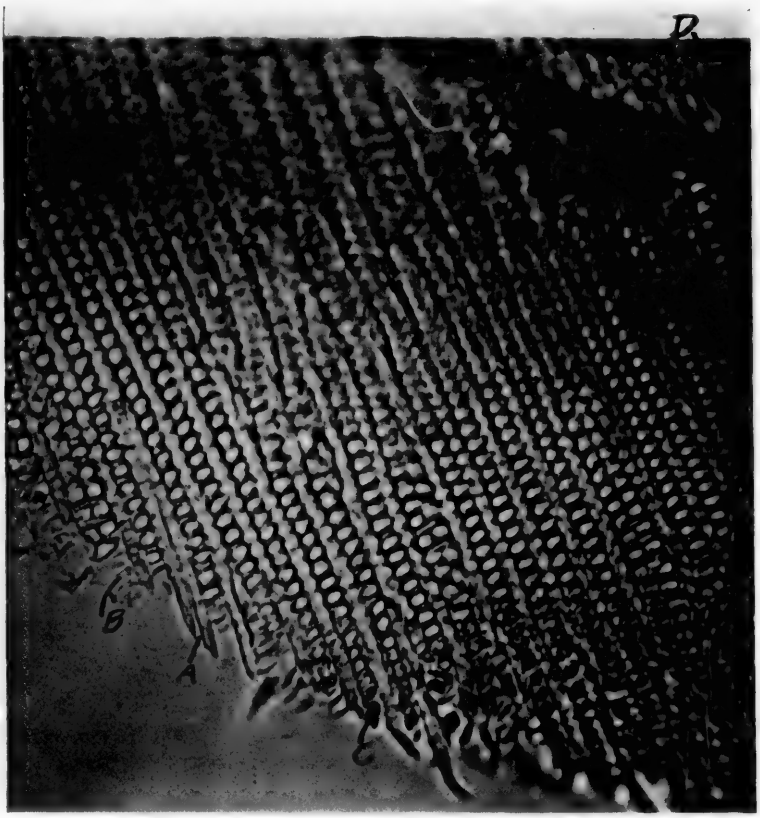

FIG. 6. Ground section through enamel layer of rat incisor. Area lies posterior to second molar; shows no evidence of crystallization, although of full width. Ganoblast layer lost at this place during grinding. *A* indicates splitting apart of material between rows of globules. *B* shows space where globule has been lost, and clear view of rung-like process of ladder-like unit of matrix. *C* points to individual globule. *D*, dento-enamel junction.

enamel. But what nature accomplishes so generally and so easily would be difficult to reproduce artificially, and in the laboratory would require perhaps high pressures and temperatures; and we have not been successful in obtaining an answer to this question. We are inclined to believe, however, that the difference between the positive and the negative crystal is one of CO_2 content.

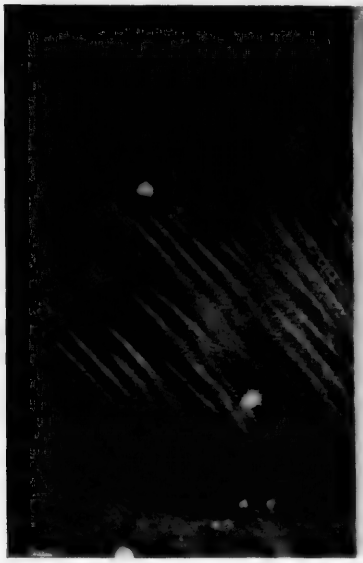

FIG. 7

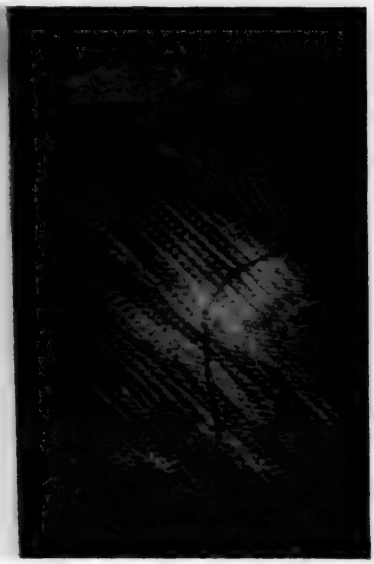

FIG. 7a

FIG. 7. Ground section through enamel-layer in area where first signs of crystallization appear. Bright lines are sides of ladder-like units, and rows of globules are between these lines. Some indication here that some of rung-like connections are crystalline. Globules show practically no birefringence at this point. (Photograph taken with polarized light.)

FIG. 7a. Same area as shown in fig. 7, but in ordinary light.

Returning to the examination of rat enamel we find that, even when the crystal phase has reached a final form in the ladder-like processes, there is slight evidence of crystals in the globules. They show but slight birefringence, and the alternation between the rows of globules and the material between them is very evident, owing to the higher birefringence of the latter. *Fig. 8* shows the transition zone described

as isotropic; the initial crystal phase to the left of this zone; and the final phase on the right. This photograph was taken in polarized light. *Fig. 8a* is the same area in normal light. We must proceed a relatively long distance from the transition zone toward the incisal edge of the enamel before evidence of the crystallization of the globules is definite. Finally, however, the rows of globules, and the ladder-like processes between them, become equally birefringent and we have the

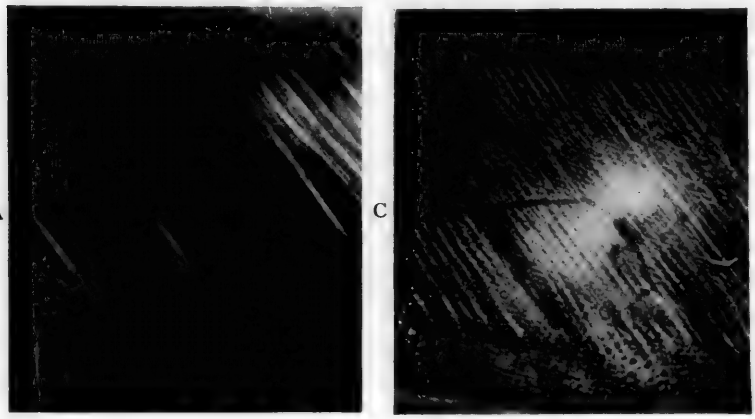

FIG. 8 FIG. 8a

FIG. 8. Ground section through enamel-layer, where transition occurs from initial stage of crystallization, showing negative sign of elongation to final crystal condition with positive sign of elongation and negative crystal sign. *A* indicates younger side of material. Selenite plate in optical system, when aligned with these bright lines, shows those on *A* side as blue, and those on *C* side as yellow, indicating change from one type of crystal to other. The dark area between known as anisotropic crystal condition. (Photograph taken with polarized light.)

FIG. 8a. Same area as shown in fig. 8, but photographed with ordinary light.

mature enamel condition. *Fig. 9* is a photograph in polarized light of completed rat enamel. On the left (root side) of this area there is still evident the greater birefringence of the material between globule rows, indicated by *S*, over the birefringence of the globules, indicated by *G*. On the right (incisal side) this difference is lost in an equal birefringence. *Fig. 9a* is a photograph of the same area with normal light.

The observations on developing enamel in the rat incisor are in gen-

eral agreement with the phases observed in previous work on human enamel (3). There it was noted that, in deciduous and young permanent teeth, there were frequent areas of enamel with crystals of a positive optical sign surrounded by material with crystals of optically negative sign; and always separating the two was an isotropic condition. On the other hand, in sound teeth secured from patients 65–70 years old, the enamel throughout its extent, as seen in thin sections,

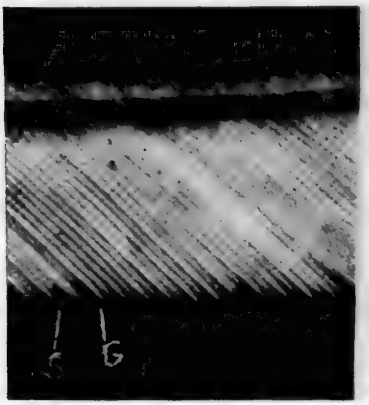

FIG. 9

FIG. 9a

FIG. 9. Ground section through rat enamel-layer in area well anterior to first molar region. Signs of crystallization evident in globules, but greater birefringence still shown on left side in material between rows of globules. Toward right (older) difference less obvious. *S* indicates material between globule rows. *G* shows row of globules. (Photograph taken with polarized light.)

FIG. 9a. Same area as shown in fig. 9, but taken with ordinary light.

was uniformly composed of crystals with optically negative sign. The condition in the younger teeth left us in doubt as to whether we might not be dealing with a retrogressive change as well as a progressive one. In the light of the undoubtedly progressive changes in the rat enamel-development, it seems logical to believe that those areas of positive crystals in young human teeth were uncompleted crystal phases, and denoted the lack of some material necessary for the production of a final phase of crystallization. As has been suggested heretofore, this is probably a condition of phosphate predominance that failed to change

to a carbonate predominance. While it has been possible with human enamel to take the negative crystal-phase and change it into the positive, by application of red heat and by immersion in acid celloidin, this rat-enamel study has shown the building up process that previously could be envisaged only from the stages in the process of destruction.

Without involving anyone excepting the author in possibly erroneous conclusions on this subject, appreciation is expressed for the kind help and criticisms of Dr. W. J. McCaughey, of the Department of Mineralogy, Ohio State University.

Summary and conclusions

(1) The phases of the process in the development of enamel crystals have been studied in the incisor tooth of the white rat.

(2) The theory of the production of the finished and calcified enamel rod, by the individual ameloblast, is not supported by the observations on rat enamel-development. The claim of a mass change subsequent to the completion of an organic matrix is supported.

(3) The individual crystal, which is the unit of structure in the completed enamel-rod of rat incisor, is placed with its unique axis at right angles with the long axis of the rod. It is an optically-negative crystal with a positive sign of elongation.

(4) In the initial stage of crystallization, the individual crystal is probably arranged in the same relation to the long axis of the rod as the final form. Since its sign of elongation is undoubtedly negative, this combination points to an optically-positive crystal. The change, from an optically-positive crystal-form through an isotropic state to a final optically-negative form, is evidence of an addition through solid solution of some material, probably a carbonate, in the process of crystal development.

(5) Since the place at which all evidence of enamel tissue disappears (in decalcified sections of rat incisor) is the place at which the final phase of crystal structure makes its appearance, it is indicated that, where ordinary decalcified sections are studied, the degree of enamel calcification may be determined by the presence or absence of residual structure in that layer, well-organized residue indicating that practically no calcification occurred.

(6) It is believed that a method of observation has been outlined

by means of which one may note minute changes in the enamel layer of experimental animals, and thereby determine more accurately what the effect of experimentally controlled variables may be on this tissue.

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MANIFESTATIONS OF VITAMIN-A DEFICIENCY IN A HUMAN TOOTH-GERM[1]

Case Report

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Numerous papers have appeared in the medical and dental literature concerning effects of systemic disease on the structures of the teeth. Congenital syphilis, rickets, and the exanthematous diseases of childhood have been most commonly mentioned as the cause of hypoplastic tooth-formation. The possible rôle of vitamin-A deficiency in the production of such defects in the teeth is the subject of the present report.

Hypoplasia, irrespective of the nature of the causative factor, occurs only in those teeth in which enamel and dentin are being laid down during the period of the disease process. In the human the permanent incisors, first molars, and cuspids, and the temporary second-molars, are most frequently involved. The lesions are bilaterally symmetrical, and occur along the incremental lines in both enamel and dentin. The subject is well reviewed and summarized in a recent report by Karnosh (1). Hypoplastic tooth-defects similar to those occurring in the human have been produced in laboratory animals by various investigators. Erdheim (2) has reported the effects of parathyroidectomy, and Schour and Van Dyke (3) of hypophysectomy, on the teeth of rats. The effects of rachitogenic diets have been observed by McCollum and his co-workers (4) on the teeth of rats; by Klein (5) on the teeth of rats and swine; and by M. Mellanby (6) on the teeth of dogs, rabbits, and rats. The effects of diets deficient in vitamin C, on the teeth of guinea pigs, have been described in detail by Höjer (7), and by Wolbach and Howe (8).

[1] Aided by a grant from the Milton Fund of Harvard University.

Changes owing to deficiency in vitamin A, in the teeth of rats, have also been reported by Wolbach and Howe (9). The rate of growth of the teeth was described as being greatly diminished or completely arrested. Histologically, atrophy of the enamel organ was noted, the highly specialized epithelium having been replaced by a narrow layer of stratified, non-keratinizing epithelium. The odontoblasts also showed marked reduction in size and loss of their normal epithelioid arrangement. During atrophy the odontoblasts continued to form an irregular, poorly calcified dentin matrix. The odontoblasts occasionally became incorporated in this matrix, giving it an appearance somewhat similar to that of bone. No studies have been published on the human tooth-germ during periods of vitamin-A deficiency. Block (10) has reported the condition of the fully erupted teeth in a series of sixty-four blind children having a history of xerophthalmia in infancy. Nineteen cases having shown evidence of advanced rickets, the tooth defects could not be considered specific of any one deficiency disease. In six cases, the teeth were normal; in twenty-four, in which the permanent molars were defective, the incisors were normal. He concluded that, in man, deficiency in vitamin A has no specific injurious effect on the formation and calcification of the teeth.

The case to be described was encountered among the routine post-mortem examinations at the Children's Hospital. During the past two years, a section of the mandible containing the permanent first-molars has been removed at autopsy from each of fifty-six cases of congenital syphilis and nutritional disturbance. Eight instances of congenital syphilis are the subject of a forthcoming report. Study of these cases, and of the literature on the subject, has made us certain that the changes in the tooth germ described in this report were not due to syphilis. There is a division of opinion as to the etiological factor in the production of hypoplastic teeth in congenital syphilis. Some investigators have held that the local presence of Treponema pallidum in the tooth follicle was responsible for the lesions observed; others, that a non-specific systemic factor associated with malnutrition was the essential influence. This subject will be discussed in a forthcoming report. The histological findings in the enamel organ in the present case are referable to a nutritional disturbance, and are not due to the general or local effects of Treponema pallidum.

Acknowledgment should be made of the constant interest and helpful criticism of Dr. S. B. Wolbach, and of the several members of his department whose coöperation have made possible this investigation.

CASE REPORT

History and examination. S. W., infant; 3½-months old; born one month prematurely. Patient, 6½ pounds (2.9 kg.) at birth; breast fed for three

FIG. 1. MESIO-BUCCAL CUSP OF PERMANENT FIRST MOLAR OF TWO-MONTHS-OLD INFANT

Structures shown are essentially normal for this age.

a, Outer enamel epithelium. *b*, Stellate reticulum. *c*, Stratum intermedium and ameloblasts. *d*, Artefact. *e*, Enamel. *f*, Dentin. *g*, Pre-dentin. Hematoxylin and eosin; ×50.

weeks; then diet changed to condensed milk and water for three weeks. Vomited on this diet; one of whole milk and dextro-maltose substituted. Continued to vomit and lose weight until time of admission to hospital at age of 3½ months. One-half teaspoonful of cod-liver oil given daily since four weeks old, and two teaspoons of orange juice since eight weeks

old. On admission to hospital: patient poorly developed and nourished. Snuffles and maculo-papular rash present. X-ray examination: fine mottling of cranial bones, and very marked, irregular, sub-periosteal new-bone formation along shafts of all long bones; also some irregular serration at

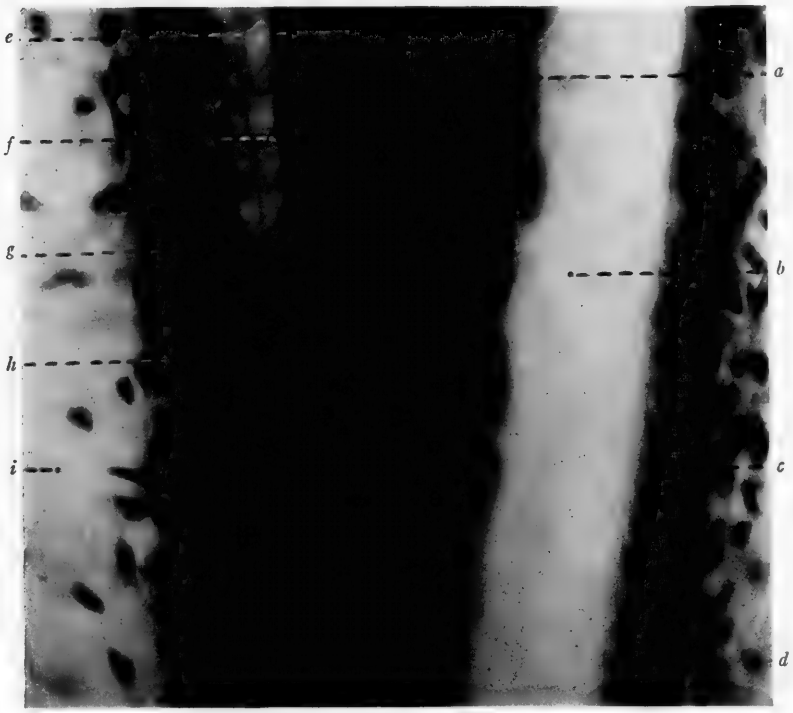

FIG. 2. HIGH MAGNIFICATION OF OUTLINED AREA IN *fig. 1*

a, Dentin. *b*, Pre-dentin. *c*, Odontoblasts. *d*, Cells of dental pulp. *e*, Enamel. *f*, Tomes' processes pulled from enamel. *g*, Ameloblasts. *h*, Stratum intermedium. *i*, Stellate reticulum. "H & E;" ×600.

metaphyseal margins. Clinical diagnosis: congenital syphilis. Patient died twenty-four hours after admission to hospital.

Post-mortem examination (A-31-13). Post mortem examination made five hours after death. Diagnosis of congenital syphilis confirmed. Spleen, liver, and pancreas fibrosed. Bones: wide layer of calcified cartilage; moderate irregularity of osteochondral junctions; and wide zone of peri-

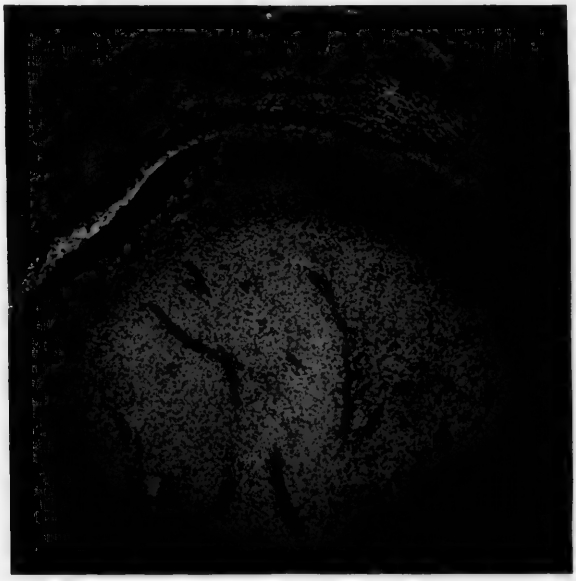

FIG. 3. VITAMIN-A DEFICIENCY CASE. TIP OF DISTO-BUCCAL CUSP OF PERMANENT FIRST-MOLAR

Atrophic enamel organ, with degenerating ameloblastic layer, well shown. Note also small cap of enamel, granular calcification of dentin ending abruptly at limits of enamel deposition, and width of pre-dentin.

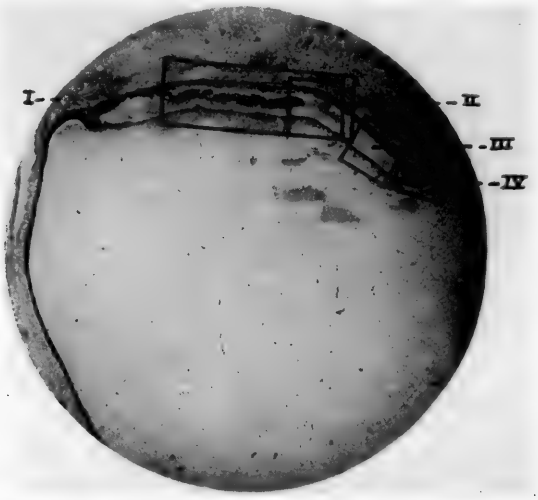

FIG. 4. MESIO-BUCCAL CUSP OF PERMANENT FIRST-MOLAR OF VITAMIN-A DEFICIENCY CASE

Section cut below tip of cusp in region lacking enamel deposit. Areas marked in squares shown in higher magnification in *figs. 5–8*.

osteal, proliferative periostitis. Bone marrow: fibrosed and infiltrated with chronic inflammatory-cells. In addition, evidence of unsuspected vitamin-A deficiency demonstrated. Epithelium of trachea, and of pelvis of kidney, replaced by keratinizing, stratified, squamous epithelium. Epithelium of enamel organ replaced by non-keratinizing, stratified epithelium.

Mandible and tooth germ. X-ray examination of section of mandible removed at autopsy: marked periostitis similar to that in other bones.

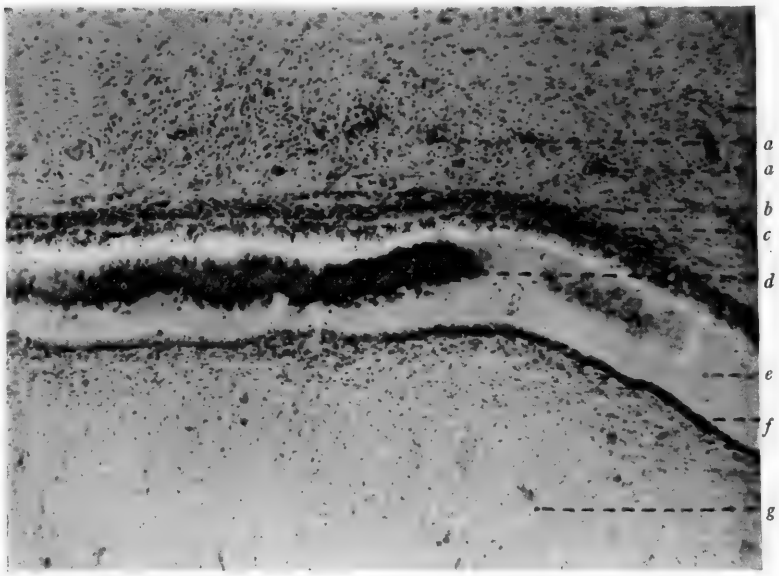

FIG. 5. AREA I OF *fig. 4*

a, Small blood vessels in loose connective-tissue above tooth germ, showing perivascular inflammatory-cell infiltration. *b*, Layer of stratified epithelial-cells replacing outer enamel-epithelium, stellate reticulum and stratum intermedium. *c*, Degenerating ameloblasts. *d*, Granular calcification of dentin. *e*, Wide pre-dentin. *f*, Odontoblasts. *g*, Dental pulp. "H & E;" ×112

Beginning calcification in mesial and buccal cusps of permanent first molar. No calcification in distal, or disto-lingual, cusp. Microscopic examination of tooth germ: presence of beginning-calcification in mesial and buccal cusps confirmed. Enamel cap small, but otherwise not unusual (*fig. 3*). Enamel organ strikingly atrophied. Ameloblasts recognizable only where enamel formation lacking—at sides of tooth germ, and between cusps on

occlusal surface (*fig. 4*). Toward center of cusp, where enamel normally present, ameloblasts degenerated—scarcely recognizable (*fig. 5*). Process started probably with appearance of clear spaces between ameloblasts. Many of cell nuclei displaced. Similar clear-spaces observed between ameloblastic layer and dentin in delicate mesh-work of cell processes, which

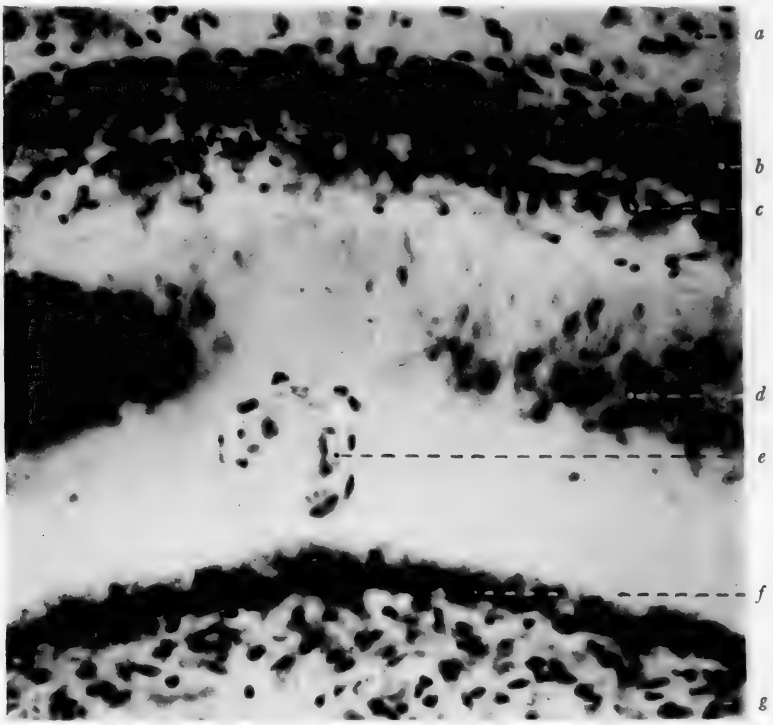

FIG. 6. AREA II OF *fig. 4*

a, Connective tissue with inflammatory cells. *b*, Layer of epithelial cells. *c*, Ameloblasts. *d*, Granular calcification of dentin. *e*, Capillary loop in pre-dentin. *f*, Odontoblasts. *g*, Dental pulp with inflammatory cells. "H & E;" ×450.

ran from ameloblasts and apparently terminated in dentin. Along meshes of cell processes were scattered fairly numerous nuclei; most, pyknotic (*fig. 6*). At sides of cusps, where dentin formation not far advanced, histological picture essentially normal. Ameloblasts assuming columnar form; nuclei situated toward base of cell; basement membrane separated amelo-

blastic layer from cells of stratum intermedium (*fig. 7*). Small area of stellate reticulum just distal to dentin formation on mesial cusp. This normally delicate structure obscured by infiltration of lymphocytes and

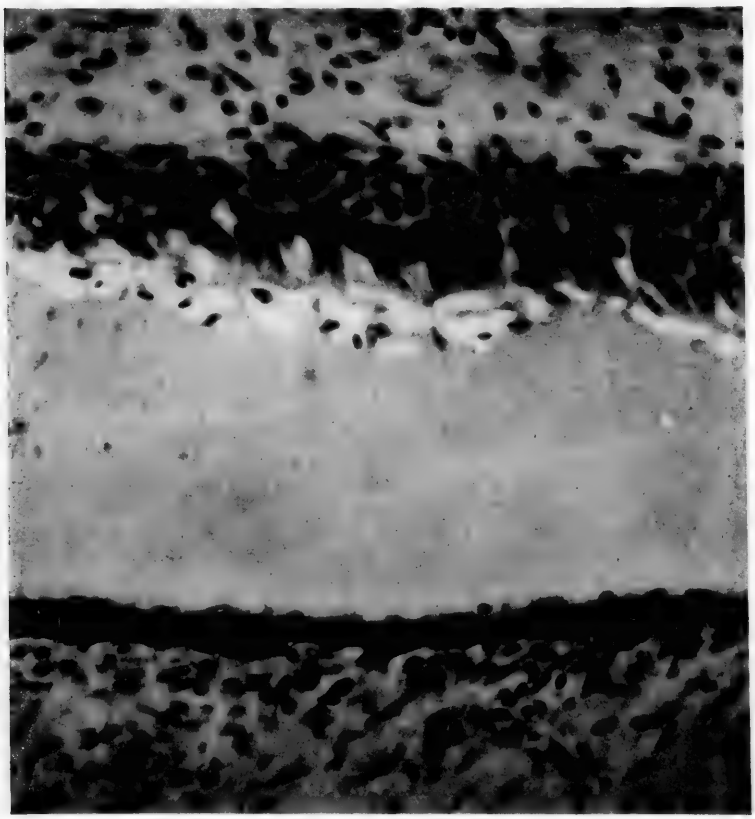

FIG. 7. AREA III OF *fig. 4*

Structures in same order as in *fig. 5*. Notice normal arrangement of ameloblasts (right). At left, instead of enamel normally present, ameloblasts here degenerate; clear staining-spaces between cells, and between ameloblastic layer and dentin. Meshwork of cell processes and pyknotic nuclei also well shown. Compare with normal in *fig. 2*. "H & E;" ×450.

large mononuclear phagocytes. Phagocytes appeared distended by homogeneous eosin-staining material. Outer enamel-epithelium: normal-appear-

ing cells clumped in large masses (*fig. 8*). Excepting this small area, stellate reticulum completely absent. Where enamel or dentin matrix present: overlying ameloblasts backed by layer of squamous epithelial cells. Dentin very defectively calcified. Abnormally small globules of calcium salts deposited only under areas where enamel was (or should normally

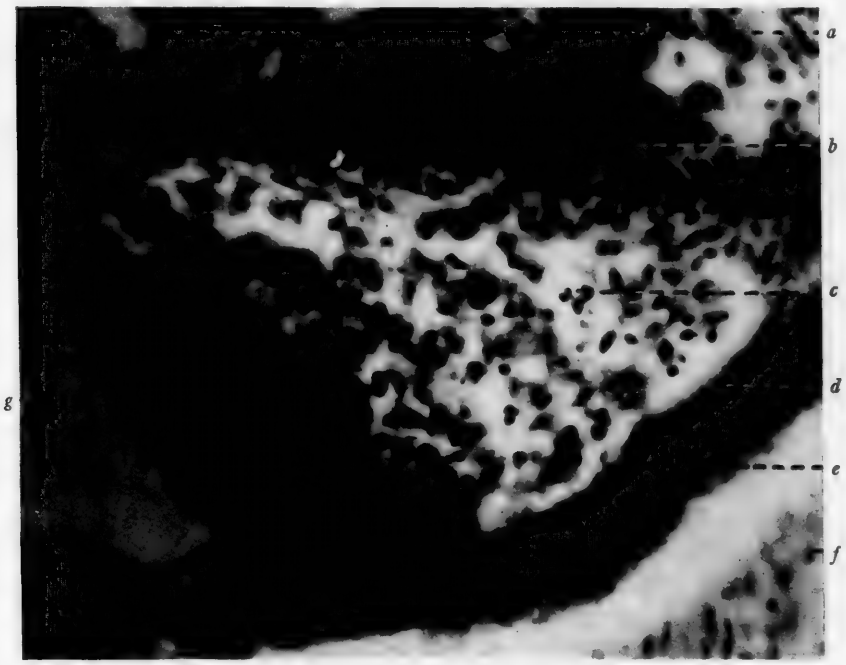

FIG. 8. AREA IV OF *fig. 4*

a, Loose connective-tissue and inflammatory cells above tooth germ. *b*, Clumped cells of outer enamel-epithelium. *c*, Stellate reticulum with inflammatory-cell infiltration. *d*, Stratum intermedium. *e*, Ameloblasts. *f*, Odontoblasts. *g*, Pre-dentin. "H & E"; ×450.

have been) deposited (*fig. 3*). Pre-dentin matrix several times normal width; in numerous areas, penetrated by small capillaries (*fig. 6*). In other places, atrophic cells of odontoblastic layer incorporated in pre-dentin. Odontoblasts reduced in size, and many nuclei pyknotic. Dental pulp: scattered, chronic, inflammatory-cell infiltration; otherwise not unusual.

COMMENT

Metaplasia of the epithelium in the trachea, pelvis of the kidney, and enamel organ, place this in a group of vitamin-A deficiency cases, which, at the Children's Hospital in Boston, are the subject of a report to be published from the Pathology Laboratory of that Hospital. The tooth germs were not examined in a previous case of keratomalacia reported by Wilson and DuBois (11). The lesions observed in the tooth germ, in the case reported in the present paper, are not characteristic of rickets. There was definite evidence of congenital syphilis in the mandible as well as in other organs and tissues of the body. Diffuse and perivascular, chronic, inflammatory-cell infiltration was observed in the dental pulp and in the loose connective-tissue about the tooth germ. There was also marked periostitis with periosteal bone proliferation in the mandible. The type of atrophy of the enamel organ and the quality of calcification of the dentin cannot, however, be explained on the basis of congenital syphilis. Atrophy of the ameloblasts, with cyst formation in the stellate reticulum and inflammatory-cell infiltration of the enamel organ of the human, have been described in cases of congenital syphilis by Cavallaro (12), Römer (13), and others. The loss of stellate reticulum, atrophy of the enamel-forming cells with little evidence of inflammation and none of necrosis, and the replacement of the highly specialized epithelium by an undifferentiated squamous type of epithelium, are collectively definite phenomena of vitamin-A deficiency.

The sequence of events in the organs affected by vitamin-A deficiency varies in individual animals of the same species. There are also variations characteristic of the species of animals already studied, i.e., rat (9), guinea pig (14), and man (11). In the rat, atrophy of the enamel organ has been described as a late manifestation of vitamin-A deficiency. If this is also true of the human, an explanation may be sought in this fact for the occurrence of normal teeth in six patients of Block's series of children who suffered from xerophthalmia during the period of tooth formation and calcification. His 64 cases were most, if not all, of the survivors of 368 cases of keratomalacia occurring in Denmark from 1912 to 1920. The cases that survived would presumably be less likely to show permanent defects as a result of late manifestations of vitamin-A deficiency. It has long been known that

a large proportion of cases of congenital syphilis do not manifest dental stigmata. The exanthematous diseases likewise leave their mark on the teeth of relatively few of the children affected. The fact that fifty-eight of Block's sixty-four xerophthalmia patients showed hypoplastic tooth-defects, only nineteen of which might be referred to rickets, does not seem to justify his conclusion that vitamin-A deficiency in man has no specific effect on the formation and calcification of the teeth.

The enamel organ of the constantly-growing incisor of the rat contains no stellate reticulum. The almost complete disappearance of this structure in this case, at a period of tooth formation when it normally constitutes by far the largest part of the enamel organ, is therefore particularly interesting. If similar changes take place in the stellate reticulum of the molar of the rat (or other animals), a study of the repair phenomena might be expected to shed some light on the function of this little understood structure. With the exception of atrophy of the stellate reticulum, the changes in the tooth germ in this case are practically identical with those described by Wolbach and Howe (9) in the tooth germ of the rat in vitamin-A deficiency.

Summary. The histological findings in the tooth germ of a $3\frac{1}{2}$-month-old infant have been described. Atrophy of the enamel organ, with cessation of enamel formation, was noted. The highly specialized enamel-forming cells, and the stellate reticulum, were replaced by a non-keratinizing layer of squamous epithelium. The dentin was defectively calcified. The pre-dentin was abnormally wide and showed capillary and cell inclusions.

Conclusion. Vitamin-A deficiency may be the cause of tooth hypoplasia in the human.

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VINCENT INFECTION: ETIOLOGY, DIAGNOSIS AND TREATMENT[1]

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INTRODUCTION

There is probably no disease in the mouth about which there is so much confusion in the mind of the average dentist as about Vincent infection. This is due in no small measure to incomplete knowledge on the subject, and to the countless contradictions which have crept into medical and dental literature regarding its etiology, communicability, classification, differential diagnosis, and treatment. As examples of these contradictions it has been said to be caused by the Vincent group of organisms and denied by others (2, 5); by one it is said to be "highly contagious" (6); by another, "mildly infectious" (8); while a third says he has "never observed a case of infection of a healthy mouth by a patient suffering from the disease" (2). Attempts at classification are also attended with contradictions, some classifying it according to its clinical manifestations, while others deny the existence of more than one type of the disease (5).

In the field of treatment most writers are agreed that oral sepsis is the chief predisposing factor in Vincent infection, and yet they disagree as to the place it should occupy in treatment, generally urging that no attempt at cleaning the teeth be made until all symptoms have been brought under control by medication. The different types of medication are almost as diverse as their advocates, although all generally agree on the value of oxidizing agents. And as if to make confusion worse confounded, the disease is referred to by a multitude of names. It is little wonder that under these conditions widespread confusion exists or that mistakes in diagnosis and treatment occur. It is too

[1] Read at a meeting of the New York Academy of Dentistry, Yale Club, New York City, January 11, 1932.

much to hope that all these contradictions can be cleared up at this time, but it may not be out of place to consider them with a view to a clearer understanding of the disease and the ways in which it may be prevented and successfully treated.

ETIOLOGY

What is the cause of Vincent infection, and what are the factors that predispose to its development? In answer to the first part of the question, it may be said that the weight of evidence at present points to the fact that the fusiform bacillus and spirochete in symbiosis are the chief etiologic factors. It is because of this relationship that diseases caused by these organisms are referred to as fusospirochetal infections. As might be expected, there is a diversity of opinion as to their relative etiologic importance, although it would seem that the spirochetes, which are believed to be the organisms that explain in many cases the difference between the acute and subacute forms of Vincent infection, are the chief offenders. Indeed, there are those who say that a laboratory diagnosis of Vincent infection can be made only when the spirochete *Treponema Vincenti* is found to be the predominating organism (9). The organisms are present, as a rule, in the mouths of most individuals, but their capacity to cause disease depends upon something more than mere presence. This being true, their presence, even in large numbers, is not necessarily diagnostic of Vincent infection, although Appleton says that he "regards the presence of these organisms in the human mouth as indicative of something wrong" (1). And that which is usually "wrong" is bad hygiene, since it creates conditions favorable to the development and growth of mouth bacteria, including the Vincent organisms.

Oral sepsis is probably the chief predisposing factor in Vincent infection, since it favors the growth of all kinds of organisms and at the same time tends to reduce local resistance. Clean, well-cared-for mouths are not highly susceptible to any form of periodontal disease. In the treatment of any of them, cleanliness is the first principle. The capacity of the Vincent organisms to cause disease depends, as in all infections, upon three things; namely, number of organisms, virulence of the organisms, and resistance of host, and in this trinity, resistance is probably the controlling factor. It is loss of resistance,

with subsequent infection by one's own mouth organisms rather than by contagion, that probably explains most cases of Vincent infection. Excessive smoking is another of the common and fashionable ways in which resistance to the Vincent organisms is impaired; it and oral sepsis being the chief factors in increasing susceptibility.

While there are those who regard the disease as highly contagious, and one that may be acquired from eating utensils, public drinking cups, and even from osculatory reciprocity, there are others who regard it as only mildly infectious and believe that it cannot be communicated to a person possessing a clean mouth (2). My own experience confirms the latter observation. That it can be conveyed from one mouth to another, and actually cause disease under conditions of bad hygiene and lowered resistance, is true; but it does not appear that the danger of such transmission is very great where proper hygiene is observed. It is my belief that most cases seen in private practice are self infected. Failure to keep the mouth clean, and excessive smoking, tend to lower resistance to the point where the organisms, already present, cause infection through multiplication or possible increase in virulency. At least I see very few cases in which the mouth is normally clean, and free from the influence of tobacco smoking, that become infected with the Vincent organisms. There are such cases, and I have seen some; but they are the exception, not the rule.

DIAGNOSIS

There are at least two clearly distinct types of Vincent infection as seen in the mouth; namely, the acute and subacute types. It is this fact that frequently leads to confusion in diagnosis and treatment. The *acute* form is the one we are all the more familiar with; not that it is more common, for it is not, but because its symptomatology is such that it can hardly be confused with any other periodontal disease. In its typical form it involves the gingivae of one or both jaws, and is characterized by a grayish slough, highly inflamed gum tissues, odor, and temperature. The subjective symptoms are pain, prostration, loss of appetite, bad taste and the like. Not all the gingivae are usually involved. Removal of the slough reveals a raw tender surface that bleeds easily. Occasionally the lips, inside of cheeks, tongue, and tonsils are involved, as indeed other parts of the body may be. Po-

tentially, Vincent infection is the most dangerous of the more or less common diseases of the mouth, sometimes having a fatal termination. Its most dangerous form is noma (7, 10).

The *subacute* type is quite different. There are no clearly defined objective or subjective symptoms as in acute Vincent, and for this reason mistakes in diagnosis are frequently made. The objective symptoms are inflamed gingivae, usually septal gingivae, that bleed on the slightest provocation. The application of a little pressure on such gingivae is sufficient to cause a distinct hemorrhage. In appearance this type of Vincent infection resembles gingivitis so closely that a differential diagnosis cannot, as a rule, be made from the objective symptoms alone. It is on the subjective symptoms that one must rely for a differential diagnosis. These are usually pain, bleeding, and bad taste, although the three are usually not present. The chief diagnostic symptom is pain. It has been said that Vincent infection is always painful (8); in typical cases this is certainly true. Therefore, given a case of inflamed gingivae without any subjective symptoms, one is usually safe in making a diagnosis of gingivitis. In saying this, however, it is realized that there are those who believe that there is a third type of Vincent infection, called chronic or potential Vincent's, in which there are few if any subjective symptoms, but which has all the ear marks of a very mild form of the subacute type. Dean says that this type of Vincent infection "may have a slow insidious onset and present slight clinical manifestations" (11).

In these "chronic or potential" cases there are usually a few septal gingivae the crests of which are abnormally red in color and which bleed freely on pressure. In these cases, almost identical clinically with the subacute type, the hygiene is usually fair; there is no generalized gingivitis and no history of pain. The absence of the latter symptom is puzzling. Inquiry in these cases usually reveals excessive smoking. Whether these cases represent an exceedingly mild form of Vincent infection I am not certain. If we adhere to the dictum that all Vincent cases are painful, as is true of the acute and subacute types, then we must rule these out. If on the other hand we agree with those who say that in every case in which there are Vincent organisms, plus the objective symptoms of a subacute type, we are dealing with a form of Vincent infection, then we are justified in so diagnosing these cases.

One thing that I have observed is that, when these cases are put on an anti-Vincent-infection treatment, they invariably respond. This I believe is good practice; admitting however that it proves nothing, for it is a treatment that can be applied to advantage to other forms of gingival inflammation, inasmuch as the first principle in the treatment of all periodontal disorders is the establishment of good hygiene.

In some recent work by Cahn (12), he describes two cases, diagnosed as chronic Vincent infection, in which he found, from sections of the gingivae, an unidentified fungus deep in the tissues, which he believes was the real cause. His description of the cases as "painful" would rule out gingivitis, and also that type of Vincent infection usually referred to as chronic or potential, as in the latter type there are no subjective symptoms. They would seem to resemble more nearly the subacute form, which is always accompanied by pain. An interesting question in this connection is whether the fungus described by Cahn is not one of the phases in the life cycle of the Vincent organisms. On this point Dean says: "The fusospirochetal organisms constantly found in oral lesions diagnosed as Vincent infection represent phases in the life cycle of a higher bacterium or microscopic plant, and more than one genus may be encountered from such a source" (13). The Deans also call attention to "an unidentified organism exhibiting cultural characteristics similar to those of the bacillus fusiformis," which are present in cases of Vincent infection, and which are believed to belong to the same biologic group (14).

TREATMENT

When we come to consider treatment, here again there is great diversity of opinion, although as already stated there is rather general agreement as to the value of oxidizing agents. What these should be, and their place in the program of treatment, is still a matter of debate. It is generally agreed that oral sepsis and tobacco-smoking act as predisposing factors in Vincent infection and that, as a first step in treatment, smoking should be prohibited and efforts made to destroy the Vincent organisms by the use of oxidizing drugs. But we are told, by some, that the teeth should not be cleaned as there is danger of spreading the infection, when as a matter of fact the mechanical cleaning of the teeth is the most effective way of ridding the mouth of the

infecting organisms and bringing about a speedy cure. This conception of treatment was put forward when Vincent infection was recognized only in its more virulent form, and when experience in its treatment was more or less limited. In the acute stage, where there is considerable sloughing and prostration, care would naturally have to be taken to prevent injury to the already infected and painful tissues. Indeed, the sensitiveness of the gingival tissues under these conditions is such that it would be impossible to clean the teeth with any degree of thoroughness. Nevertheless, the establishment of good hygiene at the earliest possible moment, consistent with the patient's comfort, is clearly one of the first principles in the treatment of Vincent infection. In making this statement it is realized that there are those who still cling to the older conception of drugs first and cleanliness afterwards, a mode of procedure that explains in many instances the long drawn out treatment so often reported in Vincent cases.

The logic of reversing this procedure must be apparent when one considers all the facts. We are dealing with anaerobic organisms, which thrive only in places of lowered oxygen tension, such as is found in gingival crevices, around partially erupted third molars, in periodontal pockets, etc. When it is further realized that many cases of Vincent infection are superimposed upon an already existing periodontoclasia with more or less deep-pocket formation, providing ideal locations for the development and growth of the Vincent organisms, the difficulty of speedily reaching these organisms by oxidizing drugs and mouth washes is self evident. Having removed them as far as is possible by mechanical means, then the use of oxidizing drugs for destroying those that remain is the logical procedure. The importance of relying on cleanliness rather than on drugs has been emphasized by Beust:

"A discussion of the microbial phase of Vincent's infection should logically consider the means for the disposal of the microbes. The great lesson taught by the microscope has not, in my estimation, been heeded by the profession. It is not the determination of variety, but the demonstration of the presence of microbes, that is important. We have learned from microscopic examinations that, in this lesion, the interdental spaces and gingival pockets are filled with a teeming mass of virus. Notwithstanding this knowledge it is common practice to handle these cases gingerly—to trust

to the defenses of the patient to ward off the onslaught. The treatment given by the average practitioner is practically useless. Superficial applications will never affect the organisms in the interior of the pockets. These concealed armies are the real offenders. If the best is to be done for the patient, then the activities of these organisms must immediately be suppressed. The hemorrhage incident to an immediate energetic treatment disperses the bacteria and their products, reduces the hyperemia, and affords almost instantaneous relief to the patient. If the pockets are evacuated and left surgically clean, the much feared spread of infection will never occur. Anyone capable of treating a pyorrheal pocket can get satisfactory results. Microörganisms that may already have entered the tissues, a condition recently depicted by Cahn (3), will fall a prey to the bodily defenses as a result of the improved tonicity of parts. The effect will be gratifying to both patient and operator (2)."

These observations by Beust I have repeatedly confirmed in an experience of more than twenty years in the treatment of Vincent infection. I do not hesitate to say that the establishment of good hygiene represents the first principle in its treatment. This should be done, as far as is consistent with the patient's comfort, at the first visit. I would especially call attention to Beust's statement, as quoted above, that the "microörganisms that may already have entered the tissues . . . . will fall a prey to the bodily defenses as a result of the improved tonicity of the parts." Inducing gingival hemorrhage in these cases, as can be done without trauma, not only raises the tonicity of the gingival tissues, but at the same time aids in ridding them of their bacterial invaders.

AUTHOR'S PROCEDURE IN TREATMENT

In outlining a procedure in treatment, which I have found to be as nearly perfect as anything in life can be, I do so realizing that other methods will also give good results. I shall refer to a method of treatment I have adopted, after trying many others that in my hands proved less satisfactory, believing that when properly employed it will give universally good results. Having made the diagnosis (and it makes no difference which type of Vincent infection I may be dealing with), a small quantity of chromic acid (5 per cent) is poured into a dappen glass, and liberally applied to the teeth and periodontal tissues. Chromic acid (H_2CrO_4) is an exceedingly important oxidizing agent in

the treatment of Vincent infection. It may be obtained by crystallization from a mixture of bichromate of potassium and sulphuric acid. Being a trioxide, it stands first in its oxygen liberating properties, a 5-per cent solution yielding about 20 per cent of available oxygen. Potassium permanganate stands second, with about 16 per cent, while sodium perborate, so popular in these cases, yields but little over 2 per cent (4). Hydrogen dioxide is another oxidizing solution, yielding about ten volumes of free oxygen (11).

Having bathed the teeth with the chromic acid, and allowed it to flow into the gingival crevices, the surfaces of the teeth are carefully polished with brush wheel or porte polishers, after which the mouth is carefully sprayed and the same procedure followed with the teeth and gums of the other jaw. In this way the chromic acid bathes the periodontal tissues for two or three minutes before being washed away, which is important. This having been done, an instrument is next passed into each gingival crevice or pocket, and its contents carefully removed, for it is in these places that the organisms occur in largest numbers. Without injury or pain, bleeding is encouraged. As already stated, this is done to rid the gingival tissues of the organisms in these tissues, where according to Dean they produce "thrombi and wedge-shaped infarctions" (10). Having completed the scaling and curettment, the gums are again bathed with the solution of chromic acid, and the teeth further polished and taped. The patient is asked to refrain from smoking during treatment. Alcohol is also interdicted. In acute cases attention should be given to diet and elimination, the diet to include fruit juices and dairy products in liberal quantities. The following mouth wash is then prescribed:

℞ Hydrogen dioxid aquae 8 ounces
Mercury bichloride 2 grains

Sig: Two teaspoonfuls in a half-glass of warm water; use every hour as a mouth wash.

In some cases this is alternated with another mouth wash composed of the following:

℞ Potassium permanganate 15 grains
Distilled water 6 ounces

Sig: A tablespoonful in a quarter-glass of warm water; use every hour as a mouth wash.

Potassium permanganate may discolor the teeth. It is, however, an excellent oxidizing agent, and occasionally gives better results than some of the other more commonly used washes. The stains can be removed by polishing the teeth. A 0.5 or 1 per cent solution of chromic acid is a most efficient mouth wash, and may be used when indicated. In very bad cases it has much to recommend it.

The frequency and number of times the patient should be seen depends upon the case. Usually from once a day to once a week, and from three to half a dozen times, will clear up most cases. Each time the patient returns to the office, the treatment outlined above is carried out, applying the chromic acid and following this with a careful prophylactic treatment. All treatment is directed to one end; namely, the establishment and maintenance of a high order of mouth hygiene with a view to reducing the Vincent organisms and promoting local resistance. It is the latter that is of first importance, for, if maintained at a high level, it is not likely to be broken down no matter how abundant the organisms may be.

With the case under control the patient is taught how to take care of the mouth, and proper brushes and dentifrices are prescribed. The following dentifrice is exceedingly useful. I ask all patients to use it for several months:

℞		
	Sodium perborate	9 drams
	Calcium carbonate precipit.	3 drams
	Powdered castile soap	30 grains
	Saccharine	2 grains
	Oil wintergreen	15 drops
	Oil peppermint	1½ drops

Sig: Use on tooth brush in cleansing teeth two or three times a day.

Unfortunately, infection by the Vincent organisms does not create immunity. Nevertheless, if the principles outlined above are carefully adhered to, the danger from reinfection is almost nil. If normal hygiene is maintained, and smoking is kept within reasonable limits, there is little danger of infection or reinfection, for in this way mouth bacteria are reduced to a minimum and the natural resistance of the periodontal tissues is promoted.

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DISCUSSION

F. C. Cady, D.D.S., Lieutenant-Commander, U. S. Public Health Service (New York City): As I listened to this paper, I was reminded of a case that was referred to our clinic yesterday. The patient, age 32, had a history of secondary syphilis, acute gonorrhea, and chronic bronchitis. We found in addition chronic pyorrhea, with a superimposed acute Vincent infection. I thought this man deserved sympathy, with all his various afflictions, and accordingly he was hospitalized for treatment.

Perhaps my experience with Vincent infection in government clinics or hospitals, extending over a period of twenty years, has been more extensive than it would have been in private practice. I recall that my first contact with Vincent infection was in 1914, at an army post in the Philippines. Peculiarly, it was all among white people of the enlisted men and officers of the service. I examined a great many native troops, and cannot recall one

case of Vincent infection in a native. My next contact with Vincent infection was in France during the war, at a base hospital where there was a great deal of the virulent type, complicated by various wounds of the face and mouth. Here we had some very unusual experiences. I believe we experimented with practically every treatment including strong escharotics, aniline dyes, and the arsenicals. We were not familiar with the secondary symptoms of Vincent infection at that time, probably because of the mobility of the troops and the fact that they apparently responded so readily to treatment. It was not until after the war that we recognized there were subacute conditions of Vincent's. My experience now is mostly with seagoing men—U. S. coast guard and the merchant marine—and of course we find a great deal of Vincent infection, particularly the sub-acute type of the disease.

I am at a loss to know just why it is that this infection is so persistent unless, as the essayist has said, "being anaerobic they dig deep." The anatomy of the dental structures perhaps works against the treatment, as there is no opportunity for physiological rest. The mouth must function no matter whether it is diseased or not. There also are the free margin of the gum, pyorrhea, pyorrhea pockets, defective fillings, and defective bridge abutments—all places that cannot be cleansed, and convenient for infection. I am not so much concerned with the curative treatment as I am with mouth hygiene and preventive treatment. Unquestionably this procedure alone will eradicate Vincent infection. Until dentists restore teeth with effective margins and with normal contact points, replace them with self-cleansing appliances, and teach their patients correct mouth hygiene, I am quite sure this very distressing infection will not be eradicated. It is not good surgical or medical practice to expect to eradicate an infection of this type with curative treatment alone. It is our experience that it is poor practice to attempt to cure the acute conditions of this disease without first scaling and polishing, and removing all defective restorations and appliances.

In many instances we had to extract teeth for patients having acute symptoms of Vincent infection. We have noticed slight untoward effects from this procedure. A man who presents with an acute pulpitis, and who is going to sea in a short time for a long period, must have the tooth removed. We do not hesitate to extract after thorough scaling and treatment with chromic-acid solution. Our procedure is to thoroughly scale a mouth during the acute stages. We do not find it necessary to wait until sloughing and the acute symptoms have disappeared. We find this procedure a great aid in shortening the length of time for clinic treatment.

It has not been our experience that smoking is a chief etiological factor in

Vincent infection, although tobacco undoubtedly is a predisposing factor by lowering the resistance of the dental tissues. Those who take good care of their mouths are rarely afflicted with Vincent infection.

I. Hirschfeld, D.D.S. (New York City): I feel that I can agree with practically everything Dr. Merritt has said, so far as my own clinical experience is concerned. Nevertheless, there are a few matters that I should like to discuss. Dr. Merritt has given a very noteworthy array of mouth washes and drugs. According to many reports, all of these have been used with marked success in the treatment of Vincent infection. In reflecting on the favorable reaction of the disease to such a variety of drugs, the thought occurred to me that, in addition to their action on the microörganisms, there might be some common factor in the process of their application, which uniformly aided in the characteristically rapid resolution of the acute symptoms. This I suspected was the mechanical and cleansing action incidental to their use. With this in mind, I proceeded, on several occasions, to treat the infection by removing gently, with instruments, the food débris and necrotic tissue, and directing the patient to rinse thoroughly and frequently with plain distilled water. While some favorable progress was made, it was not sufficient to warrant the continuance of such procedure. Nevertheless it indicated that the massaging and cleansing action are of therapeutic value. Thus I feel that the technique employed is deserving of consideration.

Regarding the number of visits the patient makes to the office: I recall that up to about eight years ago I required altogether about ten or twelve visits. With further experience, I discovered that in many cases, according to those I had occasion to see again, the treatment had not really been completed. One of the characteristics of this infection is that you can resolve the symptoms quite abruptly with just a few thorough treatments, but the disease may not be completely eradicated; at least, that has been my observation. Several were actually cured; but a large number did not come back for further treatment because they felt comfortable and had very little hemorrhage, and accordingly did not feel it necessary to pay any additional visits involving extra expenditure. Even if there should be a return of slight interproximal hemorrhage, the patient's occasional use of the mouth wash is often sufficient to prevent this subacute condition from becoming acute. To forestall the disadvantages that such an interruption in treatment entails, I changed my method of charging from a per visit to a case basis. The amount due is paid before the active treatment is completed, and the patient is then entitled to come periodically for a check-up during the remainder of the year, or until there is not the slightest evidence of a recurrence.

Dr. Merritt has pointed out that all the symptoms enumerated are not necessarily present in any one case. That is something we should always bear in mind. However, in the acute infection, at least, two symptoms are constant: hemorrhage and pain or tenderness. When the patient is unaware of the latter, it may be due to the fact that the infection is limited only to protected septal tissue, which the toothbrush bristles fail to reach. If the operator finds, on examination, that these areas are not tender to touch, the probability is that he is dealing with a different disease entity. For instance, only last week, I had a case of pregnancy gingivitis, which had been diagnosed as a Vincent infection. In the dentist's attempt at treatment, he used a strong escharotic, which burned the mouth badly. He might have arrived at the proper diagnosis had he checked up on the absence of gingival pain or tenderness. This leads me to one more practical point in the subject; namely, the atypical condition of Vincent infection, either acute or subacute, which may present a marked hypertrophy of either the papillae or the entire gingivae. This clinical picture may easily simulate entirely different pathologic entities, possibly symptoms of serious systemic disease. The differential diagnosis in these is easily made by the therapeutic test. The patient is placed on anti-Vincent treatment for about forty-eight hours; if a marked improvement is seen, this treatment is continued; if not, the dentist must look for another diagnosis.

O. J. Chase, D.D.S. (New York City): Dr. Merritt did not emphasize very strongly the question of smears in Vincent infection. I do not know whether he uses them. Of course, as Dr. Merritt said, when a number of cases are seen, the diagnosis appears apparently simple. Because of the importance of the disease and the fact that some cases terminate fatally, the question arises whether the general practitioner is justified in continuing the treatment if he does not obtain rather rapid results. I should like to speak of the possibility of leukemia in connection with Vincent infection. I was called to see a patient who was very ill, and apparently had a very acute Vincent infection. The tonsils were badly involved. A nose and throat specialist had been called; later Dr. Vaughan, together with two physicians. A blood count revealed leukemia. Patients frequently have leukemia with acute Vincent infection. When we have to deal with a disease that is so important, and may be a question of life and death, it would be wise to issue some warning to the effect that unless results are promptly obtained and the disease rapidly subsides, an oral surgeon or specialist should be consulted.

C. M. Taylor, D.D.S., Major, Dental Corps, U. S. Army (West Point, N. Y.): I should like to say something about Vincent's as an army dental surgeon. We have thousands of cases. We started when we were in France or probably before we left. I think I have tried everything that anybody has mentioned, hoping to arrive at some conclusion as to wherein one treatment was better than another, but I have come back to the prophylactic treatment as given tonight by Dr. Merritt. His paper contains everything we can do. I thought tonight I might learn something new, but, as it happens, I do exactly as Dr. Merritt described in every respect even to the mouth washes. When however I was with the First Division in France, we proceeded to Germany, and went into bivouac. Before that our cases of Vincent's were comparatively few; but after the troops went into bivouac, something caused the whole organization to contract Vincent's. I wondered at the time exactly what it was. We were able to put the worst cases in the ward. We had about fifty or sixty very bad cases, which were isolated. I had them treated with chromic acid but, in addition to that, we put them on a fresh-fruit diet, especially orange juice. It seemed that that did more than anything else for them. It is probable that, when the troops went to the front, they were so excited they didn't think about their tooth brushes. Probably the great number of cases we had was due to the fact that oral hygiene is very poor among enlisted personnel. We use 7 per cent chromic acid, and have no hesitation in so doing. Instead of trying to scale the teeth, when the men first come in, we give the whole mouth a good spraying. So far as I know, there is no detriment in doing it. I have never noticed any, at least. By this spraying, the immediate pain is overcome and they are comfortable that night. As soon as possible we start to scale the teeth and cleanse them.

While we were at Brest, France, we had a great deal of Vincent's. I tried neosalvarsan, wondering how that would affect the condition, using no mouth washes at all and no chromic acid. The neosalvarsan helped to a certain point, but after that the condition was no better unless I used a local application of chromic acid.

William B. Dunning, D.D.S., F.A.C.D. (New York City): I wish to commend the good common sense of Dr. Merritt's paper. I am not a specialist in periodontia. I attend to the average number of cases of Vincent infection that come to the general practitioner, and I fully agree with Dr. Merritt—who always comments on things in such a clear and simple way—in stressing the importance of good oral hygiene as a preventive measure against Vincent infection. The point is well made that we can get after this condi-

tion gently and effectively, but immediately, and not feel that a number of visits must be insisted upon for preparatory medication before anything can be done with instruments. We are inclined to be timid in the treatment of Vincent's, and it is helpful to be told by one who knows that we should start right in and clean up a mouth surgically, along with such therapeutic measures as may be indicated.

Charles H. Large, D.D.S. (New York City): I had an experience which I should like to relate in the hope that it may draw comment from the essayist. I took charge of a dental clinic of an institution where we had about 2500 employees, and noticed at the time that there were quite a number with Vincent infection. Most of the cases were from one department, where the girls sit at adding machines and list checks. At that time the practice was to take a bundle of checks, wet the thumb, turn over the checks and operate the adding machines; then put a rubber band around the checks and pass the bundle on to another girl, who would go through the same motions, as far as wetting the thumb and fingers were concerned; then she would pass them on to a number of other girls who would do much the same thing, making different listings. Then we also examined the drinking fountains and found that, while they used sanitary cups at the drinking fountain, it was common practice if a cup stuck in the holder, to wet the thumb and pull out the cup and the next person, finding the next cup sticking a bit, would do the same thing and pull out a cup.

Without knowing whether those things caused a spread of the infection or the disease, we immediately put a stop to each practice. We warned all the employees of the department not to put their fingers in their mouths under any conditions; also that they were not to wet the thumb, if the cup stuck in the cup holder at the drinking fountain; and we were very careful not to mention the trouble that might ensue, but we stressed the fact that it was an objectionable practice and we wished they would not do it. In order to help them in the matter, we gave them rubber thumb-stalls so it was not necessary or desirable, in fact it was almost impossible, to wet the thumb because of the objection to the rubber thumb-stall being placed in the mouth. Instead of the great number of Vincent infections we had in that department, they were reduced to about one a week.

S. E. Davenport, Jr., D.M.D. (New York City): I, too, wish to compliment Dr. Merritt upon his very clean-cut and common sense presentation, characterized by the same happy choice of phrases for which I think he is famous. There is just one point upon which I should like to speak. On

several occasions when I thought I had been successful in curing a case of Vincent infection, I found to my dismay that after a fairly short time a reinfection apparently was occurring. At first I attributed the recurrence to my own inefficiency and it may be that this was chiefly responsible. But in addition I found that in these cases there were present one or more partially erupted third molars, each covered in part by a flap of loose tissue and so tipped that a deep interproximal pocket had been formed. I was unable to conquer the Vincent infection completely and permanently until these offending teeth had been extracted and the pockets, which were undoubtedly serving as foci of reinfection, thus automatically eradicated.

F. G. Neurohr, D.D.S. (New York City): I should like to ask, in reference to the question which Dr. Davenport has brought up, whether it would make any difference if the pocket were around a third molar or around any other tooth.

Dr. Davenport: I was referring particularly to cases where the third molar was so tipped that it butted against the distal surface of the second molar in such a way that the resulting pocket was more difficult to reach and cleanse, and seemed to reinfect itself more easily than pockets about other teeth more normally placed in the jaws.

Sigmund W. A. Franken, D.D.S. (New York City): We should bear in mind that there is increasing a disease which, in the mouth, is similar to Vincent's. No matter what treatment is used, there does not seem to be any improvement and the patient rapidly declines. In these cases not only should a smear be taken, but also a differential blood count. Many of these cases are agranulocytic angina. Very frequently the only symptom the patient has is general malaise, rapidly rising temperature, and a Vincent ulcer, which will not clear up under any treatment. For these patients it is advisable to consult a physician immediately, because the mortality of the disease is from 80 to 90 per cent. Otherwise the dentist is placed in a very unfortunate position if, forty-eight hours or three days after he first sees the patient, a physician must make out a death certificate. Agranulocytic angina seems to be more prevalent than it was four or five years ago, or when Schultz[2] originally described it about nine years ago.

Dr. Merritt (in conclusion): I was greatly interested to hear Commander Cady say that he did not hesitate to extract teeth in cases of Vincent infec-

[2] Schultz, W.: Ueber eigenartige Halserkrankungen. *Deutsche med. Wochenschrift*, 1922, *48*, 1495.

tion where they were forced to do so under the conditions prevailing in his service. While I am sure no one would recommend doing this except under conditions of an emergency, it is interesting to know that it has been done under such conditions without any complications. I agree with those discussing the paper that smears should be taken at any time it is felt that smears might throw light on diagnosis. However, I believe it is true that in these cases a good clinician is more important than a bacteriologist.

That Vincent infection is sometimes associated with leukemia there is no doubt. Several cases of such association have recently been reported. The cases referred to by Dr. Large were probably due to the organism having been transmitted from one individual to another. That they can be so transmitted, and cause an infection, is proven by the fact that there are records of several epidemics of Vincent infection. It will be remembered that only two or three years ago such an epidemic prevailed in Smith College. Regarding Dr. Davenport's statement about residual infection around partially erupted third molars: there can be no doubt that such partially erupted teeth, because of the pockets around them, are ideal places for the growth and development of the Vincent organism, just as are pockets in cases of periodontoclasia.

The point that I especially want to make in my paper is that the establishment of good hygiene in the mouth at the earliest possible moment represents the first principle in the treatment of Vincent infection. When this is followed by the use of oxidizing agents, few if any cases fail to make a rapid recovery.

THE SIGNIFICANCE OF FOOD-PARTICLE SIZE IN THE ETIOLOGY OF MACROSCOPIC DENTAL DECAY IN RATS

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Introduction. Recent work by Hoppert, Webber and Canniff[2] has suggested the possibility that size of food particle plays a rôle in the etiology of macroscopical dental decay in rats. Their animals ingested the following diet (adequate): ground yellow-corn, 60 parts; whole-milk powder, 30; linseed-oil meal, 6; alfalfa meal, 3; NaCl, 1. The rats on this diet developed dental decay in the molar teeth at eight weeks of age. Some of the ground yellow-corn was then sifted through a 60-mesh sieve. The portion that passed *through* the sieve (substituted for the ground whole-corn in the diet of the first group) was fed to another series of rats, which, fed for eight weeks, did not develop dental decay. The conclusion noted by these workers, namely, that size of food particle was the causative factor in the caries incidence, was not entirely justified by the experiments, for it may readily be noted that there are many differences aside from particle size between ground corn and that part of corn which passes through a 60-mesh sieve. Nevertheless, Hoppert, Webber and Canniff have, in this manner, offered an interesting suggestion, which was subjected to further investigation in the experiments described below.

It should be noted that this paper is concerned only with *macroscopic* dental decay *in rats;* that is to say, with very obvious and easily identified holes in the molar teeth. The authors have already pointed

[1] Research Fellow supported by grants from the Research Commission of the American Dental Association, and from the following members of the Dental Staff of the Johns Hopkins Hospital: Drs. B. L. Brun, H. E. Kelsey, H. H. Street, W. H. Baish, L. D. Coriell and C. V. Matthews.

[2] Hoppert, Webber, and Canniff: *Science*: 1931, lxxiv, p. 77; *Journal of Dental Research*, 1932, xii, p. 161.

out[3] that such large cavities may be produced by at least three processes of breakdown of the crown. These three avenues of attack can be identified best by microscopic serial sections of rat mandibles and maxillae. Such examination reveals that macroscopic caries

TABLE 1

Data showing the incidence of macroscopic dental caries in two groups of rats on the same diet, in which the food particles were large for one group and small for the other group

Group I. *Diet:* Steenbock; particles large. *Composition* (per cent): yellow corn, 76; wheat gluten, 20.0; NaCl, 1.0; $CaCO_3$, 3.0—Ca, 1.2; P, 0.23. Percentage of group having caries: 64; percentage of molars carious: 20. Only *lower* molars were carious.[1] *Symbols:* *L* = left; *R* = right; *1* = first molar; *2* = second molar.

Rat (no.)	272	269	270	271	1	2	3	4	5	6	K11	K8
Age (days)	61	61	61	61	160	160	160	160	160	160	163	180
Caries	None	None	None	None	L1	L1, 2	L1, 2 R2	L1, 2 R1, 2	L1, 2 R1, 2	L1 R2	L1 R1	None

Rat (no.)	K4	K5	K13	K12	K6	K9	K10	2361	2362	2363	2364
Age (days)	190	190	163	163	190	163	163	155	155	155	155
Caries	L1, 2	L1, 2 R2	L1, 2 R1, 2	L1, 2 R1, 2	None	L2 R1, 2	L1, 2 R1, 2	L1, 2 R1, 2	L1, 2 R1, 2	L1, 2 R1, 2	L1, 2 R1, 2

Rat (no.)	2365	2366	2367	2368	2369	2370	2371	2372	2788	2789	2790
Age (days)	155	155	155	155	155	155	155	155	57	57	57
Caries	L1, 2 R1, 2	L1, 2 R1, 2	None	L1, 2 R1, 2	None	L, 2 R1, 2	L1, 2 R1, 2	None	None	None	None

Group II. *Diet:* Steenbock; pulverized to pass completely through a 60-mesh sieve. *Composition:* as for Group I. Percentage of group having caries: None.

Rat (no.)	584	585	586	587	572	548	552
Sex	M	F	M	M	F	F	F
Age	209	209	209	209	191	115	164
Caries	None	None	None	None	None	None	None

[1] The rat lesions reported here were noted exclusively in the lower teeth. This, however, is not invariably the rule, for caries of the upper molars, left and right, also occurs in rats.

may be initiated by (*a*) fracture of the molar cusp, (*b*) decay at the bottom of the molar sulci, or (*c*) interproximal caries (frequently food impaction is noted here), or (*d*) all three. The character of the caries-inducing avenues of attack is determined apparently by the

[3] Klein and McCollum: *Journal of Dental Research*, 1932, xii, p. 528; June.

properties of the diet, certain diets producing only, or largely, sulcus caries; other diets inducing a preponderance of cusp-fracture caries, etc. Details concerning the analysis of the factors regulating the three caries-inducing "paths" will be discussed in later communications.

Experiments. Thirty-four rats, at weaning, were placed on the following diet: coarsely ground whole-corn, 76 parts; wheat gluten, 20; NaCl, 1.0; $CaCO_3$, 3.0. The diet was ingested for from 57 to 190 days, when the rats were killed. A second group of seven rats, at weaning, were placed on the same ration with this exception: the entire diet had been so finely pulverized that *all of it* passed through a 60-mesh sieve. These rats ingested this food until they were 115–209 days of age, when they were killed. Distilled water was given to all the rats *ad libitum*, with iodine water once a week. The presence or absence of dental decay was determined, after death, by macroscopical examination.

Results. The caries incidence in the experimental rats is given with other relevant data in *table 1.* The rats on the diet containing coarse particles showed a dental-caries incidence of 64 percent. The rats on the same diet containing no coarse particles showed no *macroscopic* dental decay.

Conclusion. The results indicate that food-particle size (corn) plays a rôle in determining the occurrence of dental caries in rats 115–190 days of age, when the rats are given diets containing 1.2 percent of Ca and 0.23 percent of P (rachitogenic ratio).

Summary. Ingestion of a coarse diet[4] containing 1.2 percent of Ca, and 0.23 percent of P, by rats for 125–160 days from weaning, was associated with the occurrence of a high incidence of macroscopic dental decay in the lower molars.

Ingestion of the same diet, in a finely pulverized form for 85–179 days from weaning, was associated with the absence of any macroscopic dental decay in the molars of the rats so fed.

[4] "A coarse diet" containing 76 percent of yellow corn.

CHEMICAL INVESTIGATIONS OF MOTTLED ENAMEL AND BROWN STAIN

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INTRODUCTION

The associated conditions known as mottled or chalky enamel, and brown stain, have been given an increasing amount of attention in the medical and dental literature of the last fifteen years. Black (1) and McKay (2) were the first to publish careful studies of the nature of these anomalies. From their work, it appears that the chalky enamel is the primary condition. It is present at the time of eruption of the permanent teeth, and seems due to a defect in the formation of the enamel, caused by the absence of the cementing substance normally found between the enamel rods. The affected areas can be seen through the smooth glazed surface of Nasmyth's membrane as opaque, dead-white blotches, which contrast with the semi-transparent normal enamel. In these chalky areas a deposit of pigmented material may develop, so that the teeth become disfigured by yellow, brown, or black stains.

The anomaly occurs mostly in certain endemic areas, many of which are in the United States (3). It has been reported from England, Holland, Italy, Spain, Mexico, Argentina, and northern Africa. It is exceedingly common in North China. At Taiyuanfu, Anderson and Stevenson (4) observed an incidence of chalky enamel as high as 82.6 per cent, and of brown stain as high as 45.4 per cent. Little is known about the occurrence of these anomalies in the lower animals, in endemic areas or elsewhere. Williams (5) has observed them in the orang utang. Pierlé (6) claims to have produced them experimentally in rats and guinea pigs by means of diets low in calcium. McKay's (7, 8, 9) extensive studies have shown that the water supply is the factor which defines the areas where chalky enamel is endemic. A

change in the source of the water supply may be sufficient to prevent the development of the anomaly in the children from an affected area. The older literature contains many other theories brought forward to account for the origin of chalky enamel. Dick (10) and Anderson (4) proposed malnutrition as an important cause. Fossum (11) thought it arose from the febrile diseases of childhood. Grevers (12) referred it to a thyroid disturbance, or other endocrine defect. Beust (13) on the other hand denied the developmental origin of the condition, and believed that the enamel was decalcified by the direct action on the enamel of agents contained in the water of endemic regions. None of these theories were supported by adequate evidence.

McKay's studies paved the way for the next step in the solution of the cause of chalky enamel. Petrey discovered, by means of the spectrograph, the presence of fluorides in the water supply of Bauxite, Arkansas. Churchill (14) reported this discovery along with a survey of some municipal water supplies. Waters from five endemic areas all showed a fluoride content of 2 parts or more per million. Smith and Smith (15) were able to correlate the concentration of fluoride in the potable water with the existing degree of mottling in Arizona. They found that endemic mottling was universal when fluoride was present in the water supply in excess of 2.7 parts per million; occasional when the fluoride content exceeded 1.5 parts per million; and absent when the fluoride content fell below this value.

Experimental proof of the causal connection between the presence of excess of fluoride in the water supply and the occurrence of chalky enamel was furnished by Smith (16) and her co-workers. These investigators reported the experimental production in rats of an enamel defect similar to mottled human enamel, by the addition of sodium fluoride to the ration, and also by inclusion in the diet of residue from the drinking water of St. David, a community in which mottled enamel is endemic. This effect of fluorides upon the teeth of rats had been noticed before, notably by McCollum (17) and by McClure and Mitchell (18), but it had not been correlated with the analogous changes in human teeth. McClure and Mitchell made chemical studies of the bones of fluoride-fed rats. They found an increase in percentage of ash, a decrease in calcium content of the ash, and depression of the Ca:P ratio. Smith and Lantz (19), working with the

incisor teeth of fluoride-fed albino rats, found no significant differences in these values, as compared with the values for normal controls. It is well established that fluorine is deposited in the bones and other tissues of animals which have received sodium fluoride with their food (18). Determination of the fluorine content of chalky enamel found in human cases, or produced in the experimental animal, have not yet been reported.

With regard to the brown stain that frequently becomes deposited in mottled enamel: nothing definite is known as to its nature. Black (1) extracted a substance from teeth which he called "brownin," and the name has been used to designate the pigment present in the stain. Manganese has been reputed to be a constituent of the stain, but this view now seems to be discarded. Gasparrini (20) attempted to bleach the color *in situ* with various oxidizing and reducing agents, light, and electricity. Most of these results were indecisive or incomplete, but nascent oxygen combined with ozone was found to be effective. Smedley (21) succeeded in bleaching the stain by the use of hydrogen peroxide. The origin of the brown stain is equally obscure. Pierlé (6) supposed it might be derived from blood serum which infiltrated the tubules of the enamel. Wofford (22) suggested ecchymosis of blood into the forming enamel as a possible cause. McKay (2) at first believed that the pigment was laid in during development, but later (7) affirmed that the discoloration occurred after eruption of the teeth. The latter view is generally accepted. Several authors have mentioned the possibility that the pigment may be deposited from without, but none of them favor this view.

A collection of mottled and stained teeth, all of which had been extracted from Chinese subjects, facilitated a chemical examination of the enamel. We tested the possibility of a decrease in the content of calcium, and a depression of the Ca:P ratio, as might have been expected from the experimental work of McClure and Mitchell (18), and from histologic studies. Certain of the theories for the origin of brown stain were tested directly. Had the stain been due to an infiltration of blood serum into the enamel, as Pierlé suggested, the organic matter should have been increased considerably. Wofford's (22) theory of hemorrhage into the forming enamel was tested by determining the iron content.

EXPERIMENTAL

The teeth selected for analysis were dried in the electric oven for several days. The enamel was then flaked off, or separated from the dentine by drilling the latter away with a bur. The enamel was then powdered in an agate mortar, weighed, ignited in platinum, and reweighed. The ash was dissolved in hydrochloric acid, and made up to volume. It was noted that the stained surface of the enamel darkened perceptibly on heating, but the powder became pure white on ignition. *Table 1* shows the figures for a series of stained and

TABLE 1

Calcium and phosphorus contents of stained and chalky enamel

TOOTH NO.	TYPE OF TOOTH	DEFECT OF ENAMEL	VOLATILE MATTER	CALCIUM CONTENT	PHOSPHORUS CONTENT
			per cent	*per cent*	*per cent*
2	Molar, carious	Stained	—	38.0	18.2
12	Molar, carious	Stained	6.9	37.2	19.0
13	Molar, carious	Stained	5.6	37.4	18.8
14	Incisor, slightly carious	Chalky	6.8	34.6	17.2
15	Incisor, carious	Chalky	6.9	39.5	19.4
16	Molar, slightly carious	Chalky	—	37.6	17.0
17	Molar	Chalky, slight staining	5.1	42.0	20.2
18	Molar, carious	Chalky, slight staining	4.4	37.8	16.2
Average			5.95	38.0	18.25

chalky enamels analysed for organic matter, calcium, and phosphorus contents. The volatile material varied from 4.4 to 6.9 per cent. Bertz (23) has reported a similar figure for normal enamel; namely, 6.82 per cent. It is therefore clear that there is no considerable deposit of serum derivatives, or other organic material, along with the stain pigment.

Calcium was determined by Van Slyke's (24) manometric method; phosphorus, colorimetrically by the method of Fiske and Subbarow (25). The results do not differ widely from the values given in the literature for normal enamel. Bertz's (23) figures, although more

than thirty years old, are most commonly quoted. He reported 35.9 per cent calcium and 17.8 per cent phosphorus. Our analyses gave an average of 38.0 per cent calcium, and 18.25 per cent phosphorus. It is clear that these figures do not show the decrease in calcium content, and depression of Ca:P ratio found by McClure and Mitchell (18) in the bones of experimental animals. They agree rather with the results of Smith and Lantz (19), who failed to find these variations in the teeth of rats.

TABLE 2

Iron content of enamel

TOOTH NUMBER	DESCRIPTION	STAINING	IRON CONTENT MG. PER CENT
	Brown-stained		
1	Molar	Slight	4.60
2	Molar, carious	Moderate	3.34
3	Incisor	Moderate	2.46
4	Incisor	Deep	1.76
5	Molar	Deep	2.31
6	Molar, carious	Deep	3.20
Average			2.94
	Unstained		
7	Incisor	None	2.53
8	Bicuspid	None	2.73
9	Bicuspid	None	2.14
10	Bicuspid, carious	None	2.51
11	Molar, carious	None	2.57
Average			2.50

Among the possible substances which might cause the brown stain, iron is one of the first which one would naturally consider, whether one conceived of it as derived from the blood, or from external sources. Normal enamel was tested, and found to contain iron. It was therefore necessary to make quantitative determinations of the amount of iron in specimens of brown-stained enamel, and compare the results with similar analyses of normal enamel. The specimens of enamel were prepared as above, and determinations of the iron content made according to the method of Kennedy (26). The thiocyanate color can

be developed directly, because no pyrophosphate is formed by ignition of the tertiary phosphates, of which the enamel is composed. The results are tabulated in *Table 2*. The average iron content of normal enamel was found to be 2.50 mg. per cent. We have not been able to find any previous analyses in the literature. The specimens of stained enamel contained an average of 2.94 mg. per cent. The difference in these averages seems unimportant. The iron content of stained enamel is less constant than that of normal specimens, but this may have little significance. It is clear that iron, and its precursor hemoglobin, are not responsible for the brown stain.

CONCLUSIONS

1. The amount of organic matter in stained and mottled enamel is within normal limits. We may assume that there is no extensive deposition of serum derivatives along with the stain.

2. The calcium and phosphorus contents of stained and mottled enamel do not show any consistent deviation from the normal values. These results do not support the decalcification theory.

3. The brown stain is not due to iron, and is therefore probably not derived from hemoglobin. Its behavior on ignition suggests that it is organic in nature.

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THE FINAL REPORT OF THE COMMITTEE ON THE COSTS OF MEDICAL CARE

Comment on a Misrepresentation of Conditions in the Practice of Dentistry

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The *Final Report of the Committee on the Costs of Medical Care* was made public at a "National Conference on the Costs of Medical Care," at the New York Academy of Medicine, November 29, 1932. The general nature of this report, and of its main parts, is now well known. The majority report contains a few allusions to dentistry. On page 112 of the majority report, appended to some "recommendations for the organization of complete medical services," including "utilization of subsidiary personnel"—(a) nursing attendants and visiting housekeepers, and (b) nurse-midwives—there is a footnote consisting of the following remarkable statement:

"*Additional statement by committee members.*—We believe that the following additional recommendation [*sic*] should be made by the Committee [as a whole]:

"c. We commend the *growing tendency in the practice of dentistry* toward a division of labor in which *a dentist who is also a physician* assumes larger responsibilities for the diagnosis and treatment of conditions arising from or related to the teeth, while *much of the routine performed by the dentist in the past* is delegated to *dental hygienists and other technicians* working under *his* direction.—Morris L. Cooke, Haven Emerson, Mrs. Walter McNab Miller, Alfred Owre, William J. Schieffelin, John Sundwall, C.-E. A. Winslow." [No italic in original.]

Those who endorsed this footnote were among the sponsors of the majority report, and, as "members" of the "National Conference" mentioned above, were listed on the official program of the Conference as follows:

Morris L. Cooke—Consulting Engineer in Management; Director, Germantown Hospital, Philadelphia, Pa.

Haven Emerson—Professor of Public Health Administration, College of Physicians and Surgeons, Columbia University, New York City.

Mrs. Walter McNab Miller—Chairman of Health Committee, National Council of Women, New York City.

Alfred Owre—Dean, School of Dental and Oral Surgery, Columbia University, New York City.

William J. Schieffelin—President, Druggists' Supply Corporation, New York City.

John A. Sundwall—Professor of Hygiene and Public Health, University of Michigan, Ann Arbor, Michigan.

C.-E. A. Winslow—Professor, Public Health, Yale University, New Haven, Conn. [Chairman of the Executive Committee of the Committee on the Costs of Medical Care].

When, on the day the report was made public, the present writer read the quoted footnote, his information indicated that the alleged "growing tendency" was an invention. Steps were promptly taken to correct or to confirm this conclusion. On December 1, 1932, and daily thereafter for about a week, letters setting forth the facts above related were sent to a total of ninety-one leading dentists in nearly all the states in the United States and the provinces in Canada, with a request for answers to these questions:

(1) Is there, in your community, any tendency or any *growing tendency* in the practice of dentistry toward a division of labor by which *dentists who are also physicians* assume larger responsibilities for the diagnosis and treatment of conditions arising from or related to the teeth?

(2) Is any of the routine work of *intra-oral practice*, involving *diagnosis*, *treatment*, or *operation* (excepting cleansing of the mouth and teeth, and polishing the latter), as "performed by the dentist in the past," now being delegated, in your community, to dental hygienists, and to other technicians, working under the direction of the said *dentists who are also physicians*?

Answers were received from all but three of those to whom these inquiries had been addressed. *The replies were unanimous in confirming my understanding that the alleged "tendency" does not exist in dental practice anywhere in Canada or the United States.*

On December 13, 1932, an effort was begun, in correspondence with

one of the seven sponsors of the quoted footnote, to obtain, if possible, a correction of the inaccurate and misleading statement contained therein. The correspondence, which affects the public welfare, is reproduced below, where the names of the responding member of the group that signed the footnote, and of the responding general officer of the Committee, are not disclosed.

CORRESPONDENCE WITH A MEMBER OF THE COMMITTEE ON THE COSTS OF MEDICAL CARE WHO, WITH SIX OTHERS, ENDORSED THE INACCURATE AND MISLEADING STATEMENT IN THE FOOTNOTE ON PP. 112–13 OF THE COMMITTEE'S MAJORITY REPORT

1. December 13, 1932: Letter from the present author to the member of the Committee

You may recall that my interest in research in dentistry was active before your pupil . . . . conducted Ph.D. work with me in this field nearly . . . . years ago. Six years have passed since my report on dental education was presented to the Carnegie Foundation. Lately, I have been giving most of my time to editing the *Journal of Dental Research* and promoting the development of the International Association for Dental Research. These relationships have made it desirable for me to keep myself informed about conditions in dental practice and in medical practice.

On the day the *Final Report of the Committee on the Costs of Medical Care* was formally released (Nov. 29), I examined a copy for references to dentistry. I read with surprise this footnote on pp. 112–113 [quoted as given above]. Unfortunately, this footnote seriously misrepresents the situation in a number of important particulars (A-D):

(A) In the footnote, your group of seven "commend" a "growing tendency in the practice of dentistry" that not only does not exist anywhere in the United States, but which, in the main, the dental statutes of all the states actually prohibit. A portion of what you "commend" would be unlawful wherever in this country it might occur.

(B) The number of dental practitioners who have both medical and dental degrees, *and as dentists are also licensed to practise medicine*, is smaller now (when both degrees must be *earned*) than it was, say, forty years ago, when recognized medical schools, for a small fee, accepted the dental degree (D.D.S. or D.M.D.) as equivalent for all

or nearly all the work for the M.D. degree, and awarded the M.D. degree a year—or *often only a week*—after the candidate received the dental degree. Page 1609 of the issue of the *Journal of the American Dental Association* for September, 1932, presents recent comment on the easy way in which dentist-physicians used to be multiplied under medical auspices and registered for the practice of dentistry and medicine.

(C) During the past decade there has been no increase in the number of dentist-physicians who "assume larger responsibilities" in dental practice—who treat "conditions [anywhere in the body?] arising from or related to the teeth." There has also been no change in the function of the dental hygienists, who may not legally *do* any more substantially than the first dental-hygienist law permitted (Connecticut, 1907). The existing dental law in . . . . [Conn.], which has been generally followed in other statutes, reads, on this matter, as follows (Sec. 16; Public Statutes, Sec. 2813):

"Dental hygienists may remove calcareous deposits, accretions and stains from the exposed surfaces of the teeth and directly beneath the free margin of the gums, *but shall not perform any other operation* on the teeth, mouth, or diseased tissue of the mouth, or attempt to make diagnosis of the same. They may operate in any office of any licensed dentist, or in any public or private institution under the general supervision of a licensed dentist. *The dental commission may suspend or revoke the license of any licensed dentist who shall permit any dental hygienist operating under his supervision to perform any operation other than that permitted under the provisions of this section*" [as specified above]. [No italic in original.]

(D) The context of the quoted footnote indicates that the words, "much of the routine performed by the dentist in the past," refer to routine *intra-oral* practice. But routine *intra-oral* practice cannot legally be delegated, anywhere in this country, to any unlicensed assistants either by dentists or physicians. If what you "commend" as a "growing tendency" in this respect actually occurred, it would express a cumulative increase in illegal dental practice by technicians. In order that such a tendency might develop and grow *lawfully*, the statutes would have to be changed to permit it. In some cities there has been an increase in the amount of illegal dental practice by tech-

nicians for pecuniary gain, not for the benefit of any patient. Although the statutes prohibit intra-oral examination, replacement, or prescription by any one not licensed to do so, here and there some unworthy dentists have delegated certain mechanical restorations to technicians. Ethical dentists have been exposing, and strenuously endeavoring to stop, all such illegal and undesirable procedures. The major portion of the current prosecutions for illegal dental practice in the United States consists of cases arising from illegal acts in dental laboratories and by dental technicians. I assume your group has not intended to commend this situation. Dental practice, in this respect, is very similar to medical practice—the crooks have not yet been completely eliminated from either.

The conditions your group "commend" in the footnote on pp. 112–113 are those of the well-known "master-servant" plan. In 1921–22, during the progress of the Carnegie Foundation's study of dental education, the conditions of this plan were thoroughly discussed with physicians and dentists, and then in 1922 almost unanimously rejected by them. This plan was formally resurrected in 1930 by the dean of one of the dental schools, in his published annual report to the president of his university. The plan was then again promptly and almost unanimously rejected in all parts of the country. These reactions represent existing realities. Therefore, it cannot be truthfully said that there has been any *growing tendency* in dental practice to adopt this plan or anything resembling it.

The report refers, in a footnote on page 140, to "experimental medico-dental programs" that a number of universities are "working on," and names Rochester, Yale, and Columbia as preeminent in this relation. At the *University of Rochester*, in 1920, it was announced that the School of Medicine and Dentistry, then soon to be created, would aim to make dentistry a specialty of medical practice, and some of the graduates would be physician-dentists. This plan has evolved by successive stages, from abandonment of these purposes—without graduation of any physician-dentists—to the attempt, since 1929, to do something more useful; namely, to give dentists special opportunity to conduct *graduate work as dentists*. Of the eight "Rockefeller Fellows in Dentistry" now at Rochester [1931–32], none is a candidate for the M.D. degree. The experiment in progress at *Yale Univer-*

sity constitutes an important test of one of the elements in the plan of reorganization of dental education proposed in my report to the Carnegie Foundation ("*Bulletin No. 19*," page 19), and which in the spring of 1928, with the approval of the Carnegie Corporation, was among the matters brought formally, in conferences with President Vincent and Director Pearce, to the attention of the Rockefeller Foundation. The experiment at Yale, begun in 1929, does not represent a "growing tendency," especially when the concurrent reversal at Rochester is noted with it. I understand that no dentist-physician has yet been graduated at Yale. No one knows whether the dentist-physicians to be graduated at Yale will engage in statutory medical practice, or in statutory dental practice, or both. But it seems certain that they will not engage in a practice that would direct technicians to violate the laws of their respective states. At *Columbia University* no one is required to obtain the M.D. degree for admission to, or graduation from, the dental school. An examination of the Announcement of the Medical School at Columbia for 1932–33 shows that there was no person having a dental degree in any of the four classes in 1931–32. The conditions of legal practice of dentistry in New York are not determined by any dental school. The basic conditions of legal practice of dentistry in the state of New York are specified in the dental statute. The number of dentist-physicians in the state of New York, who, licensed to practise dentistry, engage in medical practice, or vice-versa, or both, is practically negligible. The results of the experiments at Columbia, Rochester, and Yale do not justify your group's statement in the footnote on pp. 112–113.

An additional surprise, in evaluating the footnote on pp. 112–113 as sponsored by your group and quoted above, is a contradictory remark in a statement of opinion (by practically the same group) in the footnote on page 141, and reading as follows:

> "A variety of technicians and assistants *could be* trained, in shorter courses [than the 'course' for the M.D. 'oral specialist'], for most of the routine work *now* performed *in dentistry*." [No italic in original.]

In this footnote there is no suggestion that "most of the routine work *now* performed in dentistry" should be done under the *direction* of "dentists who are also physicians." But assuming it was your ex-

pectation that this would be inferred, then the words "could be" seem to imply an admission that technicians and assistants *are not* engaged in the routine work *now* being performed in *intra-oral* dental practice, and that the "growing tendency" alleged in the footnote, on pp. 112–113, is not in fact *growing*.

Believing that these opinions [as summarized above] reflect the truth in the situation, but wishing to test them directly before sending you this letter, I promptly forwarded, to many widely distributed correspondents, letters that included the following [two] questions [as quoted above]. From these correspondents, in all parts of the country, the answers already received are unanimously "No" to each question; and most correspondents add that the procedure outlined in question (2) is illegal in their states. Several correspondents, referring to the footnote (pp. 112–113) quoted above, add this opinion (using the words of one of them):

"If any of the members [of the group of sponsors of the statement in the footnote on pp. 112–113] have observed dental operations being performed in the human mouth [by dental hygienists or technicians], e.g., taking of impressions or the adjustment of dentures, such persons should report, to the proper legal authorities, all such unlawful acts so that the best interests of the public may be conserved."

The reaction to your Committee's report will not be made favorable anywhere by misstatements of fact, or by misrepresentation. It is obviously your personal desire to avoid both. No one would wish to impair the freedom of the members of the Committee to express their convictions and opinions. But a responsible report should be strictly accurate in all statements of alleged fact, and should not bear untrue implications or false witness. If, in the footnote on pp. 112–113, the first line after "c" had stated: We *believe* that, in the practice of dentistry, there *should be* a division of," etc., the opinion thus expressed would be evaluated as an *opinion*. The quoted footnote includes, however, a distortion of existing conditions and facts, and is palpable special propaganda for a plan *that had been currently rejected*. I feel that there should be a prompt and authoritative correction of the inaccuracies in the footnote on pp. 112–113. As that footnote stands, and until formally corrected, it will tend to distract attention from the

constructive features of the report. Some of my correspondents express this view (quoting one of them):

"I am sorry to learn that the *Final Report of the Committee on the Costs of Medical Care* carries such an inaccurate and misleading statement. The uninformed reader will be misled, and the informed reader will question the authenticity of other statements in the report."

The seven sponsors of the quoted footnote are accredited to the states of Connecticut (1), Michigan (1), New York (4), and Pennsylvania (1). Correspondence with officials in each of these four states, where the sponsors have had special opportunity to *ascertain for themselves* the facts in the situation, indicates that the division of labor to which the footnote on pp. 112–113 refers, and the "growing tendency" there alleged, do not exist, and would be illegal, in any of these four states.

The replies from the correspondents mentioned above, to the questions there quoted, are being assembled for publication in the February issue of the *Journal of Dental Research* (our next bi-monthly issue), where this situation will be reviewed.[1] We should be glad to publish simultaneously, in the *Journal*, any statement you and your associates might wish to make. Correction by your group, elsewhere and much earlier, however, would seem to be desirable.

This letter is addressed to you as one with whom, through . . . . since . . . . I have had a bond of scientific interest in dental research. The letter is forwarded in the *hope (1) that you yourself will promptly test what I have written;* also that (2) you will publicly correct anything in the footnote that may be a misstatement of fact, or publicly restate more accurately what the signatory group might have intended to say. To serve your convenience copies of this letter will be forwarded to the others in the group of seven named on page 113 of the report. Copies will also be sent to other members of the general committee.

[1] The replies of the correspondents, emphatically and unanimously "No" to each of the two questions quoted above, are so completely in the negative and so uniform in their insistence that the alleged "growing tendency" not only does not exist but could not occur *lawfully* in their states and provinces, that publication of the details of the statements would be redundant.

In conclusion, I ask special attention to the fact that, in this lengthy presentation, I have not suggested that any conviction or opinion should be modified in any way. I have merely denied the accuracy of some allegations of fact, which I believe you have personally sponsored in the erroneous belief that they are neither false nor misleading, but wholly true. If your group's statement in the quoted footnote (pp. 112–113) is correct, then I must be very much mistaken. If that should be the case, I would wish to present an apology for writing this letter; and in the *Journal of Dental Research* would help to emphasize the correctness, and therefore the importance, of the statements herein protested.

2. December 15, 1932: Letter from the member of the Committee to the present author

It was a pleasure to hear from you again as I remember most pleasantly our earlier contacts in . . . .

I know of course that the problem of the relation of medicine to dentistry on the one hand and to laboratory techniques on the other is an exceedingly complex one, but it is one of great importance and one in which we have been deeply interested here at . . . .

It is always difficult for persons of different viewpoints to agree as to social trends. We all I suppose tend to read our own desires into what we observe. When we framed the footnote to which you refer we had in mind experience abroad as well as in this country, and of course we were thinking of ultimate results far ahead of immediate legal situations. The real question is whether the tendency is a sound one or not. If it is sound we shall have as you say to change the laws. I can not honestly see that any modification of our statement is necessary. We may or may not have been mistaken in our estimate of trends, but in any case the vital matter is what ought to be done, not merely what has already been done.

3. December 19, 1932: Letter from the present author to the member of the Committee

Your favor of the 15th has been received. Your expressed feeling that you "can not honestly see that any modification of our [your] statement is necessary" is apparently conclusive. You have presum-

ably noted all the various matters to which I alluded in my letter of the 13th as showing that the statement in the footnote on pp. 112–113 of your report is not correct. Your answer implies that you have concluded that I am mistaken. I concede that I may be wrong, but as yet I am unable to think so.

I expect to comment publicly on the report, as a layman. I wish to avoid mistakes. I ought not to trouble you further, after receiving your response to my first letter on this subject, but I need more enlightenment (and I believe you will be disposed to respond) regarding the matters included in the following two questions, which are stated briefly for the sake of directness and simplicity, and numbered 1 and 2, and the parts lettered (a) and (b), to make the mechanics of a response easy and convenient:

1. Where in the United States and Canada did you understand, when you endorsed the footnote on pp. 112–113 of your report, that there is a "*growing tendency*" IN THE PRACTICE OF DENTISTRY toward a division of labor in which

(a) *dentists* WHO ARE ALSO PHYSICIANS assume larger responsibilities for the diagnosis and treatment of conditions (presumably in all parts of the body) arising from or related to the teeth; while

(b) much of the *routine* performed by the *dentist* in the past (including the mouth, of course) is delegated to dental hygienists and *other technicians* working under the direction of the *dentists* WHO ARE ALSO PHYSICIANS?

2. In which countries other than Canada and the United States did you understand, in sponsoring the said footnote, that there is a "growing tendency" IN THE PRACTICE OF DENTISTRY for

(a) *dentists* WHO ARE ALSO PHYSICIANS to do what is stated in 1, (a), above; and for

(b) hygienists and technicians to do what is stated in 1, (b), above?

If I am mistaken about the accuracy of the footnote on pp. 112–113, my error can be made very clear by your answers to these two questions. If, in your response, you would simply name the provinces and states in Canada and the United States, and the countries outside of North America, in which the alleged growing tendency was *known or believed by you personally* to be in progress when you sponsored the footnote, further details that I might need to satisfy myself of the completeness

of my error, and as to the correctness of the information you were given and on which you relied, would be available from other sources. This plan is intended to serve your convenience in replying. I should like to get this matter straight, and hope you can conveniently give me the names of the states, provinces, and countries in which the alleged "growing tendency" is known by you personally to exist.

In my former letter I stated, in effect, that I had not referred to matters of opinion; did not oppose my own opinion against any other opinion; and did not raise any questions as to what should be done to promote health service for all the people. The latter is a very important matter, to which I have already made public references (copy of publication enclosed).[2] In my letter to you I referred, however, only to the allegation of fact, in the footnote on pp. 112–113, as to a "growing tendency" in the direction there indicated, which allegation of fact is, I believe, inaccurate and misleading, but which I am confident you sponsored in good faith on the basis of representations to you that you assumed were true.

I note with special interest your remark that if the "growing tendency" mentioned in the footnote on pp. 112–113 is sound, *the laws will have to be changed to make it legal.* Do you not, in this remark, say, in effect, that the "growing tendency" your group commends is a "growing tendency" among *untrained and unlicensed* persons to violate the practice acts? If this is the "tendency" your group commends, as a basis on which improved dental practice might be evolved, would it not be desirable to make that fact public? Why not *openly* encourage this violation of the laws, if this "growing tendency" is commendable? In the footnote on pp. 112–113 you say the tendency you commend is proceeding under the direction of *dentists* WHO ARE ALSO PHYSICIANS. My information as to the recent prosecutions for illegal dental practice does not indicate that there is any growing tendency anywhere among *dentists who are also physicians* to "direct" technicians in illegal *dental* practice.

As those who sponsored the footnote are among the majority who endorse the main report, the reaction to that footnote will tend also to be a reaction to the majority report. Is it wise to allow a matter

[2] Gies: Progress in health service; *Bulletin of the Medical College of Virginia*, 1930, xxvii (whole number); Nov.

of this kind to stand on a side-track unmodified and open to the serious objection that an inaccurate or misleading statement has been sponsored by a large proportion of those who present the majority report? Where or what will be the gain for any cause or any person?

I hope you will give this matter further consideration, so that the footnote may be so modified, or extended, as to make it accurate. If your footnote stated your *belief* that the practice of dentistry should be developed in the manner you *think* best, such an expression of *opinion* would be received as an opinion. A statement alleging the occurrence of a condition that does not exist, however, is in no sense inspiring, and cannot be influential as an element of persuasion after its inherent untruthfulness becomes widely known.

4. January 3, 1933: Letter from the member of the Committee to the present author

Our viewpoint seems so different that I can not conceive further argument to be particularly valuable, but I may say in answer to your questions that we had in mind first of all the tendencies in the field of medical [*sic*] practice manifest at New Haven, Columbia and elsewhere, even though those tendencies are manifest only in the thought of a few leaders; also the very definite tendencies in actual practice in Austria, Italy and Czechoslovakia.

5. January 6, 1933: Letter from the present author to the member of the Committee

I thank you for your response, which indicates the nature and the extent of the evidence you had when the footnote about the "growing tendency" was endorsed.

My letters to you on this subject, as I have repeatedly stated, were efforts to ascertain the basis for a statement of alleged fact in a public document, not to present an opinion or to disagree with an opinion. My "viewpoint" was solely that of one who wishes to know why the truth was perverted in a particular instance of public presentation.

Your letter indicates that so far as North America is concerned, the "growing tendency" mentioned in the footnote on pp. 112–13 of your report exists not *in the practice of dentistry* as alleged in the footnote, but "*only* [!] in the *thought* [!] of a *few* [!] leaders."

You refer to "very definite tendencies in actual practice in Austria, Italy and Czechoslovakia." Here, too, the *allegations of fact* in your footnote and the *actual facts* do not agree. Take Austria, for example, as to which I have conveniently at hand the following authoritative statement, by an officer of the University of Vienna, as published in the proceedings of the last general meeting of the International Association for Dental Research (*Journal of Dental Research*, 1932, xii, pp. 508–09; June):

"The new regulations for the instruction of dentists were inaugurated in Austria two years ago. Dentistry in Austria has always been considered a part of medicine, and originally only physicians were entitled to practise dentistry. However, there was no legal requirement for a special training, just as there has been, and is, none in other medical specialties. But since dental technicians have increasingly abused their rights in the practice of dentistry, this condition has been terminated by the passage of a law granting certain rights to the dental technicians during a period of transition, but preventing further increase of practitioners who are not physicians. At the same time the use of the title 'Zahnarzt,' dental physician, was made subject to the completion of a prescribed course and the passing of an examination. This gave a final legal foundation in Austria to the conception that dentistry shall be practised only by full physicians." (The remainder of the statement describes the curriculum by which *dentistry will be made the most highly educated specialty of medical practice in Austria.*)

From this authoritative statement it may be seen that the "growing tendency" in the practice of dentistry in Austria is just the reverse of that alleged in the footnote in your report.

Many believe, and I share the opinion, that you and others who sponsored the allegations of fact in the footnote on pp. 112–13 were misled by misstatements made to you. There is a wide-spread opinion as to just how and why this was accomplished.

I brought this matter to your attention, immediately [shortly] after reading the footnote, in the hope that you and your associates would *reëxamine the evidence* on which your allegation of fact was based, and that, if you found your statement unwarranted, you would, as responsible agents in avowed public service, take suitable measures promptly to *modify the footnote so that it would be both truthful and useful.*

Your admissions—in your first letter, to the effect that if the alleged

tendency is sound the statutes will have to be changed to make it legal, and in your second, that, so far as North America is concerned, the alleged growing tendency exists "*only* in the *thought* of a *few* leaders"—are sufficient, it seems to me, to suggest the urgency of very material revision of the footnote. I hope your own conscientiousness will not be suspended in this matter, which relates to public education, public judgment, and public action; and also affects the good faith of yourself and associates. I shall not trouble you any further.

CORRESPONDENCE WITH A GENERAL OFFICER OF THE COMMITTEE ON THE COSTS OF MEDICAL CARE

6. December 14, 1932: Letter from the present author to the general officer of the Committee

The enclosure is a copy of a letter I have forwarded [December 13, 1932] to . . . . and the [other] members of the group of seven who sponsored the remarkable statement in the footnote on pp. 112–113 of the *Final Report of the Committee on the Costs of Medical Care*. The replies from many dental correspondents in all parts of the country express astonishment that anything so grossly untrue could have appeared in the report. Instead of stating a *belief* that a certain tendency *would be* desirable, the sponsors of the footnote misrepresent the situation by alleging that the tendency they favor is "growing," when in reality the said tendency does not exist.

I suggest that you investigate the accuracy of what I state in the enclosure [letter in section 1, above]; and, if the comment is warranted, that early public correction be made accordingly.

7. January 12, 1933: Letter from the present author to the general officer of the Committee

I send you enclosed a copy of the correspondence between . . . . and myself on the truthfulness of the footnote on pp. 112–13 of the *Final Report of the Committee on the Costs of Medical Care*. From this correspondence you will note . . . . admissions (a) that the footnote does not apply to dental practice in the United States or Canada; (b) that, if the stated "growing tendency" were sound, the statutes would have to be changed to make it lawful; and (c) that the said

tendency exists not *in the practice of dentistry*, as alleged in the footnote, but "*only* in the *thought* of a *few* leaders."

I respectfully and formally suggest again that *truthfulness in public education*, and *fairness to the dental profession*, require that proper steps be taken, by or for the Committee, to correct or reject the inaccurate and misleading statement in the footnote on pp. 112–13 of the report.

My first letter to you on this subject was dated December 14, 1932. You did not reply.

8. January 16, 1933: Letter from the general officer of the Committee to the present author

I have your letter of January 12th with copies of letters to . . . . and his replies.

Our policy in preparing the committee's final report was to permit any member or group of members opportunity to make dissenting statements on any part of the report with which they did not agree, or additional statements dealing with subject matter not included in the report. Since, without this privilege, members of the committee faced the alternative of endorsing the report just as it was submitted by an editorial committee, it seemed important to assure members complete freedom in the preparation and publication of such footnotes. It has been made clear, we believe, that each of these various statements represents the judgment of only the person or persons signing them.

In the particular instance referred to in your letter, a footnote was prepared to which 7 of our 48 members subscribed. The officers of the committee and the other members obviously have assumed no responsibility for it. If it were in the body of the report, it would of course be quite a different matter. Since it is a statement to which, obviously, the committee as a whole does not agree, it would seem to us superfluous to make a statement to that effect. The committee as a whole, and its officers as such, do not support any of the dissenting statements.

The officers of the committee of course might invite the attention of members signing dissenting statements to any apparent inaccuracies that may be called to our attention. In this case, we should be glad

to send to the 6 signers in addition to . . . . any memorandum on the subject you may wish to prepare. We do not see, however, how we can take away from our members the right to publish over their signature as footnotes any statement they desire to make.

We thank you for going into the matter so carefully for us in your various letters. If the statement in question were in the body of the report, we should feel obliged, and should desire very definitely, to go into the matter with you carefully.

9. January 19, 1933: Letter from the present author to the general officer of the Committee

I thank you for your letter of the 16th, postmarked "Washington, D.C.4, January 18, 7 p.m., 1933." I note your statement that the footnote on pp. 112–13 of the *Final Report of the Committee on the Costs of Medical Care* is beyond the jurisdiction of the Committee as a whole. I understood that dissenting *opinions* were respectfully included in footnotes. There can be no criticism of such a desirable policy. I did not assume, however, that *untruthful allegations of fact* were given the same respectful attention. It seemed probable that the Committee would not permit its publications to be used as floats for untruths.

I had hoped that [the copies of] my correspondence with . . . . would suggest to you a suitable way to bring about reëxamination of the *foundations of the allegations of fact* in the footnote, and that this reëxamination would assure prompt correction of the obvious misrepresentations. I believed the Committee as a whole had both power and inclination to eliminate misstatements of fact from any and all portions of its public representations.

A copy of your letter and of this reply will be sent to the seven members of the Committee who sponsored the untruthful statements in the footnote on pp. 112–13.

MEMORANDUM SENT TO EACH OF THE SEVEN SPONSORS OF THE FOOTNOTE UNDER DISCUSSION

10. January 20, 1933: "Memorandum for the seven members of the Committee on the Costs of Medical Care who sponsored the footnote on pp. 112–13, of the Final Report, on conditions in the practice of dentistry"

Copies of correspondence with . . . . [a general officer of the Committee] are enclosed [copies of all the other letters having previously been forwarded].

In a statement in the next issue of the *Journal of Dental Research* regarding the outcome of the correspondence with . . . . (whose name will not be mentioned as the author of the letters in reply to mine), a summary of general conclusions will appear at the end. This summary is submitted, below, for the correction, on the printer's proof, of any inaccuracy that you might find in it and wish to indicate.

SUMMARY OF GENERAL CONCLUSIONS[3]

1. The footnote on pp. 112–13 of the *Final Report of the Committee on the Costs of Medical Care* misrepresents conditions in the practice of dentistry. The misrepresentations have been indicated to the seven sponsors of the footnote and to . . . . [a general officer of the Committee].

2. It has been admitted, by one of the seven sponsors of the footnote, that (a) if the "growing tendency" therein alleged were "sound," the statutes would have to be changed to make the said tendency lawful; and that (b) this "growing tendency" exists not *in the practice of dentistry*, as the footnote asserts, but "*only* in the *thought* of a *few* leaders."

3. One of the seven sponsors of the footnote, although making the admissions noted in conclusion 2, has stated that he "can not honestly see that any modification of our [their] statement is necessary."

4. The Committee on the Costs of Medical Care as a whole (i.e., majority), and its officers as such, do not support the statement in the footnote on pp. 112–13, nor any other statement not included in the text of the majority report.

5. All the members of the general committee have been free to publish dissent in the *Final Report*. This freedom included liberty to misstate facts.

6. The Committee as a whole has no power to correct the untruths in the footnote on pp. 112–13. This power resides only in the group of seven sponsors of that footnote.

7. In a letter dated December 13, 1932, addressed to one of the members of this group of seven (copies of which were sent to the other six), it was stated that "this situation will be reviewed" in "the Febru-

[3] This summary was included in the memorandum as forwarded, on January 20, 1933, to each of the seven sponsors of the footnote under discussion.

ary issue of the *Journal of Dental Research.*" It was also stated in this connection: "We should be glad to publish simultaneously, in the *Journal*, any statement you and your associates might wish to make. Correction by your group, elsewhere and much earlier, however, would seem to be desirable." The *Journal* has not been authorized to publish a correction (February 18, 1933).